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# Viruses of Equine and of St. Louis Encephalitis in Relationship to Human Infections in California 1937-1938

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DURING the summer and autumn of 1937, physicians in California reported a number of cases of acute encephalitis, some with a stormy course and others less violent. There were 40 deaths. Most of the cases occurred in the two large Central California Valley regions with the majority from Fresno and Tulare Counties. Fresno reported 13 fatalities, while a milder form, usually with uneventful recovery, occurred in Tulare. During the same seasonal period in 1938 a similar type of disease was recorded showing both the severe and the milder forms. Fresno and Tulare were again affected, with additional patients from Kern County at the extreme southern end of the San Joaquin Valley.

During 1937 brain material was received from 3 fatal cases but although numerous mice and a number of monkeys were inoculated, no virus was recovered. Sera were then collected through the coöperation of the Fresno and Tulare County Health Departments from many of the recovered patients in the lower San Joaquin Valley districts where the outbreak was the most severe. Sera were also obtained in connection with the State Department of Public Health from other regions in California where some form of encephalitis had been reported. The clinical history of this group[1] has been reported, so this paper will not bear upon that aspect.

It was found that about 56 per cent of the recovered patients in the Fresno and Tulare areas had neutralizing antibodies for the virus of St. Louis encephalitis,[2] while they were negative for the Japanese B virus and that of lymphocytic choriomeningitis (l.c.m.).*

* These viruses were kindly sent as follows: The Japanese B strain from Dr. S. Kashara of the Kitasato Institute, the St. Louis virus from Dr. L. T. Webster of the Rockefeller Institute, and that of the l.c.m. strain from Dr. C. Armstrong of the National Institute of Health.

Since many of these cases showed clinical pictures similar to those described for the St. Louis type of encephalitis, it was assumed up to and through part of the summer of 1938 that the etiological agents were identical even though no virus had been directly isolated. However, the percentage of positive neutralization tests given seemed much lower than those reported by other workers for sera of recovered cases after the St. Louis outbreak. Wooley and Armstrong [3] obtained 94 per cent positive sera from 39 convalescent patients; Webster, Fite, and Clow [4] 82.5 per cent from 29, and later Muckenfuss, Smadel, and Moore [5] found 69 per cent definitely positive and 9 per cent doubtfully positive of 126 sera tested from recovered encephalitic patients in the vicinity of St. Louis.

This discrepancy was not easily accounted for at the time and it was not until the autumn of 1938 that it was explained by recovery of the western strain of equine encephalomyelitic virus from the brain of a child at Fresno.[6] This was followed by isolation of the same type of virus from the blood of a man in Tulare.[7] With the knowledge that another neurotropic virus could be a responsible agent in this region additional studies were undertaken to determine the extent of the disease in reported encephalitic areas and to ascertain whether a differentiation could be made.

### NEUTRALIZATION TESTS WITH HUMAN SERA

During the autumn of 1937 and the first half of 1938, sera were collected from recovered encephalitic patients in the Central California Valley areas exclusive of Kern County, and from a few coastal towns. These sera were mainly from Fresno and Tulare Counties and were tested for antiviral substances against the l.c.m., the St. Louis, and the Japanese viruses, respectively. A certain number were also used against the poliomyelitic virus. After recovery of the human equine strains, tests were made against the new, very virulent Br variety with all the old sera available from these areas and with more sera collected later in 1938 from Kern and Fresno Counties.

Protection experiments for the encephalitic viruses were performed as follows: Equal parts of serum and the appropriate dilutions of each virus were mixed and held overnight in the refrigerator before inoculating 0.03 cc. intracerebrally into mice. After preliminary titrations and stabilizing of each virus, it was found that under the conditions given the following dilutions were the most advantageous for each type, 2 being employed for each strain and from 3 to 4 mice per serum for each dilution of virus. The dilutions for the encephalitic viruses were made from 10 per cent brain suspensions in a mixture of equal parts of broth (pH 7.5) and sterile distilled water. They were 1–10,000 and 1–50,000 for the St. Louis strain, 1–50,000 and 1–100,000 for the Japanese B, 1–100 and 1–250 for the l.c.m., and 1–1,000,000 and 1–5,000,000 for the Br human equine virus. The original western equine (California) strain was used in the dilutions of 1–10,000 and 1–100,000. Known negative and positive sera were always included with each series. Equal parts of serum and a 1–25 dilution of the 5 per cent poliomyelitic virus were also mixed and kept overnight in the cold before inoculating 1.5 cc. intracerebrally into rhesus monkeys.

Forty-nine, or 47.5 per cent, of 103 sera of the encephalitic group had protective antibodies against the St. Louis strain, although 10 were only weakly positive as shown in Table I. A serum was considered to give a positive neutralization if all the mice employed remained alive for the two dilutions, and weakly positive if they died with

TABLE I

*Neutralization Tests on Sera of Encephalitic Cases* *

| County | No. Cases Tested | Strains of Virus: Western Equine Virus (Br Strain) | | No. Cases Tested | Strains of Virus: St. Louis Encephalitis Virus | | No. Cases Tested | Positive for Both Types of Virus | |
|---|---|---|---|---|---|---|---|---|---|
| Fresno 1936–38 | 19 | 3 ⊕<br>9 + | | 23 | 1 ⊕<br>9 + | | 20 | 7 | |
| | | 12 | (63.1%) + | | 10 | (43.4%) + | | 7 | (35%) + |
| Kern 1938 | 26 | 1 ⊕<br>8 + | | 26 | 5 ⊕<br>8 + | | 26 | 5 | |
| | | 9 | (34.2%) + | | 13 | (50.0%) + | | 5 | (19.2%) + |
| Tulare 1937–38 | 14 | 1 ⊕<br>4 + | | 22 | 2 ⊕<br>12 + | | 14 | 4 | |
| | | 5 | (35.7%) + | | 14 | (63.67%) + | | 4 | (28.5%) + |
| San Joaquin 1937–38 | 4 | 2 + | | 7 | 3 + | | | | |
| | | 2 | (50%) + | | 3 | (42%) + | | | |
| Other Central Valley areas 1937–38 | 9 | 1 ⊕<br>3 + | | 11 | 1 ⊕<br>5 + | | 9 | 2 | |
| | | 4 | (44.4%) + | | 6 | (54.4%) + | | 2 | (22.2%) + |
| Coastal Areas 1937–38 | 14 | 0 | | 14 | 1 ⊕<br>2 + | | | | |
| | | | | | 3 | (21.4%) + | | | |
| Total No. | 86 | 32 | (37.0%) + | 103 | 49 | (47.5%) + | 69 | 18 | (26%) + |

* ⊕ = Weakly positive neutralization
\+ = Positive neutralization
0 = No neutralization

the strong dilution and survived the weaker. They were negative if all animals died.

Seventy-one of the 103 sera were from cases in the 3 counties of Kern, Tulare, and Fresno, in the lower San Joaquin Valley where the majority of the encephalitic reports occurred; 37 of the 71 sera, or 52.1 per cent, gave some degree of positive protection for the St. Louis virus, the largest number coming from Tulare County and the next from Kern County. There were 2 positive cases and 1 weakly positive from the coastal counties.

Of 86 sera tested from individual cases, 32, or 37.0 per cent, gave either a weakly or a strongly positive neutralization against the Br strain of equine virus: 59 of these came from the 3 designated counties with 26, or 44 per cent, positive. These figures undoubtedly would have been higher if material had been obtained from all the patients showing neurotropic symptoms. Unfortunately many of the earlier cases disappeared and blood could not be obtained later when the importance of the equine virus for man was ascertained.

There were 6 positive neutralizations with sera from other areas in the more central or the northern parts of the Valley regions and none from the coastal districts (Table I).

That so many sera showed protection tests for these two viruses was probably of some diagnostic significance, but just how much reliance can be placed in such findings may be questioned when on further analysis it was found that a number of sera were positive to both the St. Louis and the equine strains. Of 69 sera tested against the two types, 18, or 26 per cent, showed some degree of neutralization for both (Table I).

To add further to the uncertainty in regard to the value of the neutralization test in these neurotropic cases, 34 sera of the earlier encephalitic series,

which did not include the Kern County group, were tested for antiviral substances against the virus of poliomyelitis; 26, or 78.7 per cent, of these were positive but only 4 to this virus alone. Four were positive only to the St. Louis strain while 12 had antibodies for both viruses. Thirteen of these sera remained and later 2 were found to neutralize the equine strain. There was sufficient evidence, therefore, that, given a certain district, the human serum could harbor antiviral substances for a variety of neurotropic viruses.

From 1934 to 1937 many cases of central nervous system disturbances were reported by physicians from both Kern and Tulare Counties, mainly recorded as poliomyelitis. Many of these people showed only a transient muscular weakness and recovered without residual paralysis. Fresh serum was obtained from 14 of these individuals in Kern County who had been diagnosed as having poliomyelitis in 1934 to 1937 respectively. Five were positive for the St. Louis virus, 6 for the equine, and 3 for both. Taken by years, one of the 4 sera in the 1934 group was positive for the St. Louis strain and none for the equine. The 2 in the 1935 group showed some neutralization for both viruses, while of the 4 in 1936, 1 serum neutralized the equine and 2 were positive for the St. Louis type. In the 1937 series 3 of the 4 sera reacted to the equine and none to the St. Louis strain.

Some old sera were available from a poliomyelitic group in Tulare County who had been bled in 1936 and 1937, respectively. One person showed clinical symptoms in 1934 and another in 1937, but the majority were ill during 1935 and 1936. Sera from 23 of these individuals were tested in 1938 against the St. Louis virus and 11 (43.4 per cent) showed a positive neutralization, as given in Table III. Sixteen out of 20 (80.0 per cent) neutralized the virus of poliomyelitis, 8 being positive to it alone, and 6 positive to both the latter and the St. Louis strain (Table II). Only 13 of these sera remained that could be tested later against the Br equine virus. One showed a weakly positive reaction while all the others were negative. Most of the sera had been kept for 2 years in the refrigerator, so that it was interesting to note that the antiviral substances to the St. Louis

TABLE II

*Neutralization Tests on Different Groups of Individuals*

| *A.* | *Normal Individuals* | | | | *B.* | *Cases Diagnosed as Poliomyelitis* | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | *Neutralization Test* | | | | | *Neutralization Test* | | | |
| *Type* | *No. Tested* | *Equine Virus (Br)* | *St. Louis Virus* | *Equine and St. Louis* | *Region* | *No. Tested* | *Equine Virus (Br)* | *No. Tested* | *St. Louis Virus* | *Equine and St. Louis* |
| Normal non-contacts | 26 | 0 | 2 + (7.6%) | | Tulare County | 13 | 1 + (7.6%) | 23 | 11 + (43.4%) | 1 + (4.3%) |
| Normal contacts | 26 | 0 | 0 | | Kern County | 14 | 6 + (42.8%) | 14 | 5 + (35.7%) | 3 + (21.4%) |
| Normal contacts in Tulare County | 11 | 1 + (9.0%) | 6 + (54.5%) | 2 + to both (18.1%) | Bay areas | 20 | 0 | 23 | 0 | |
| Total No. | 63 | 1 + (1.5%) | 8 + (12.6%) | 2 + to both (3.0%) | Total No. | 47 | 7 + (14.8%) | 60 | 16 + (26.0%) | 4 + to both (6.6%) |

+ = Positive neutralization test
0 = No neutralization

strain still remained. None of the people with sera positive to the St. Louis virus showed any residual paralysis and all had the onset since 1934. Of the negative cases 5 had some paralysis but had generally recovered. One in the group diagnosed in 1923 had residual paralysis and his serum did not neutralize the St. Louis strain.

In order to determine to what extent the antiviral substances for these viruses might be present among groups of people such as normal contacts, normal non-contacts, and recovered poliomyelitic patients from other than the Central Valley areas, various people were bled and their sera tested for antibodies.

In a group of recovered poliomyelitic cases from around the Bay Region, 23 sera failed to neutralize the St. Louis virus while 20 were negative to the Br equine strain (Table III). Most of the convalescents showed some form of residual paralysis, although 4 or 5 were apparently normal.

Of 26 normal non-contacts (Table III) including nurses, doctors, and laboratory workers in the Bay Region, none gave any neutralization for the equine strain. One was weakly and another strongly positive for the St. Louis virus. The former was a dentist in close contact with many people and the latter had lived in Kern County before training as a nurse in San Francisco.

A similar number of sera were tested from a group of normal individuals, doctors and nurses in the Valley areas who had had some contact with human encephalitic cases during the past few years, including also a group of veterinarians who had histories of previous contact with sick horses. All were negative to both viruses.

Another small group was included from Tulare County. These people had all been associated in some way with S, the man from whose blood the equine virus had been recovered. Although one of the 11 sera was positive to the latter strain, 6 showed neutralization for the St. Louis virus, indicating contact to the latter among the general population in this region. No history was obtained from the person positive to the equine strain.

To summarize, it would appear that of a total of 63 apparently normal individuals as far as encephalitis was concerned, 8 had neutralizing antibodies for the St. Louis virus and 1 for the equine. All but 1 of the 9 had lived in the San Joaquin Valley. Of 47 tested for the equine virus, and 60 for the St. Louis strain among groups of people diagnosed as having had poliomyelitis, the only positive sera for either virus were from those residing in the San Joaquin Valley.

To confirm further the neutralization experiments on certain of the sera that were positive to the Br or new human equine strain, the tests were repeated using the original western equine (Californian) virus. Fourteen of the 21 sera, 16 of which are given in Table IV, were also strongly positive to this strain, 4 others were only weakly positive, although they had been stronger with the Br strain, while 3 were negative. The latter had reacted only very slightly to the Br virus so the amount of the antibodies could not have been very appreciable. Six sera negative to the human strain were also negative to the Californian variety. On the whole, however, the relationship between the two types of virus seemed fairly well established by this cross-neutralization with sera from cases having a history of some disturbance of the central nervous system.

No protective substances for the l.c.m. and the Japanese B viruses, respectively, were demonstrated in any of the sera of the 1937 series. These tests were discontinued for the group collected later.

## TABLE III

*Cases Positive to Western Equine Encephalomyelitis Either by the Presence of Virus or by the Neutralization Test*

| *Name* | *County* | *Location* | *Date of Onset* | *Period from Onset to Bleeding* | *Neutralizations* | | | *Age in Years* | *Main Symptoms* | *Results* | *Remarks* |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | *Equine (Br)* | *Equine (Calif.)* | *St. Louis* | | | | |
| L.M. | Kern | Bakersfield | 7-22-35 | 3 yrs. 2 mos. | weakly<br>+ | | + | 27 | headache, dizziness, muscle weakness | recovered | Native of California |
| K.M. | " | " | 7-28-35 | 3 yrs. 2 mos. | + | | weakly<br>+ | 26 | headache, vertigo, fever | recovered | Native of California |
| J.D. | " | " | Oct., 1937 | 1 yr. | + | | 0 | 11 | headache, general malaise | recovered | Native of California |
| I.MacP. | " | " | Dec., 1937 | 10 mos. | weakly<br>+ | | 0 | 43 | | recovered | Came from Montana 5 years ago |
| L.M. | " | " | 7-18-38 | 2 mos. | + | | 0 | 1 | nausea, vomiting, stiff neck, drowsiness, muscle spasms | improved | |
| C.S. | " | " | 7-7-37 | 1 yr. 3 mos. | + | slt.<br>+ | 0 | 17 | headache, fever, vomiting, drowsiness, muscle weakness | recovered | Born in Oklahoma |
| E.D. | " | Farm near Bakersfield | 7-22-38 | 26 days | + | + | weakly<br>+ | 26 | headache, fever, drowsiness | recovered | |
| B.W. | " | Buttonwillow | 7-27-38 | 21 days<br>2 mos. | +<br>+ | | 0 | 1 | fever, drowsiness, convulsions | constant convulsions, improved | Parents from Oklahoma |
| H.B. | " | Buttonwillow | 9-13-38 | 1 week<br>1 month | +<br>+ | +<br>+ | weakly +<br>weakly + | 17 | headache, vomiting, stiff neck, drowsiness | recovered | |
| J.B. | " | Kern Lake | 8-2-38 | 7 weeks | + | + | 0 | 4 | headache, fever, vomiting, drowsiness | recovered | Oklahoma in 1936 |
| J.R. | " | Edison Cabins | 8-30-38 | 3 weeks<br>2 mos. | +<br>+ | +<br>+ | 0<br>0 | 5 mos. | fever, irritable, muscle spasms, eyes affected | residuals, improved | Lived in a tank house |
| C.R. | " | Poso | Aug., 1936 and Aug., 1938 | 2 mos.<br>10 weeks | +<br>+ | weakly<br>+ | +<br>+ | 24 | double vision, nervous, irritable, drowsiness | nervous but recovered | Supposed to have had St. Louis type in Missouri 1936 |
| R.T. | " | Delano | 10-21-38 | 5 days<br>3 mos. | 0<br>+ | | +<br>+ | 28 | headache, fever, drowsiness, generalized muscle tenderness, stiff neck | recovered | |
| D.W. | " | Arvin | 8-9-38 | 5 weeks<br>1 mo. 11 days | +<br>+ | +<br>+ | 0<br>0 | 20 | headache, vomiting, stiff neck, nausea, lethargy | recovered | Lived on a farm |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| J.B. | Fresno | Fresno | 8-25-38 | 5 days | | | 20 mos. | fever, irritability, rigidity of extremities, spasms of foot muscles, coma | died in 5 days | Virus of equine enceph. found in brain |
| G.L. | " | " | 7-27-38 | 8 days | weakly + | + | + | 13 | headache, dizziness, vomiting | recovered | |
| | | | | 22 days | + | + | + | | | |
| D.P. | " | 5 miles from Fresno | 8-1-37 | 1 yr. | + | | 0 | 4 | headache, vomiting, semi-comatose, irrational, general spasticity | residuals | |
| | | | | 1 yr. 2 mos. | + | + | 0 | | | |
| M.R. | " | Out from Fresno | 10-13-38 | 15 days | + | | | 21 | headache, vomiting, chills, sore throat, weakness of extremities | recovered | |
| | | | | 25 days | + | + | weakly + | | | |
| H.L. | " | Fresno | 8-2-38 | 2½ mos. | weakly + | 0 | + | 50 | chills, fever, headache, drowsiness | recovered | Lived near the slaughter house |
| J.H. | " | Kerman | 9-22-38 | 12 days | + | | 0 | 17 | drowsiness, muscular pain, rigid extremities, sore throat | recovered | |
| J.G. | " | Kerman | 8-6-37 | 2 mos. | + | weakly + | 0 | 66 | headache, lethargy, stiff neck | recovered but weak | Serum kept over 1 year |
| F.H. | " | Selma | 7-29-38 | 1 yr. 3 wks. | + | | + | 30 | vomiting, headache, severe stiff neck, lethargy | recovered | |
| | | | | 1 yr. 5 mos. | weakly + | weakly + | + | | | | |
| A.K. | " | Sangar | 8-29-36 | 1 yr. 2 mos. | + | | + | 33 | headache, fever, drowsiness | recovered | |
| | | | | 2 yrs. | weakly + | | + | | | | |
| C.T | " | Sangar | 7-27-38 | 8 days | 0 | | 0 | 35 | stiff neck, muscle weakness, comatose, fever | recovered | |
| | | | | 22 days | + | | + | | | | |
| E.W. | " | " | 9-27-38 | 2 mos. | weakly + | | 0 | 16 | headache, vomiting, fever, malaise, lethargic and very drowsy | recovered | Worked on a farm, many mosquitoes present |
| | | | | 1 mo. | + | + | 0 | | | | |
| C.S. | " | Del Rey | 8-21-37 | 1½ mos. | + | | 0 | 46 | headache, fever, drowsiness | recovered | |
| | | | | 1 yr. 2 mos. | + | + | 0 | | | | |
| Mrs.E. | Tulare | Rural | 7-9-37 | 3½ mos. | weakly + | | 0 | 38 | headache, fever, unconscious, lethargy | recovered | |
| A.J. | " | Strathmore | 8-24-37 | 3¼ mos. | + | | + | 60 | headache, drowsiness, fever, unconscious | recovered | |
| J.H. | " | " | 9-15-37 | 3 mos. | + | | + | 52 | headache, lethargy, generalized weakness | recovered | |
| | | | | 1 yr. 1 mo. | + | + | + | | | | |
| R.McD. | " | Terra Bella | 8-20-37 | 4 mos. | | | + | 28 | headache, fever, stiff neck, lethargy, spasticity | recovered | |
| | | | | 1 yr. | + | | + | | | | |
| V.S. | " | Kaweaha River | 7-30-38 | 7 days | | | | | headache, malaise, drowsiness, stiff neck, in coma | died in 7 days | Virus in the blood |

### PERSISTENCE OF ANTIVIRAL SUBSTANCES

It is interesting to note that so far, with one exception, the antiviral substances to the Br equine strain appeared only in sera from individuals having had some definite disturbance of the central nervous system, either a recognizable encephalitis or some intermediate form difficult to distinguish from poliomyelitis. The one exception was in a person who had been in contact with a known case of human equine encephalomyelitis and lived in an apparently infected area, but whose earlier history was not ascertained.

On the other hand, antibodies for the St. Louis virus were found not only in those people with some neurological disturbance but from a certain number of apparently normal individuals, indicating perhaps a difference in the route of infection for the two viruses.

Owing to the distance between the source of material and the laboratory facilities, collection of the sera during the early part of the disease was not often achieved, so that one is left in doubt as to which virus might have been the etiological agent in many cases where the antibodies were positive for both types. In 4 instances, however, serum was obtained in 5 to 8 days and in 1 or 2 weeks after entrance to the hospital, and then again after recovery (Table III). Protective substances for the equine strain were either absent or very weakly positive at first, becoming stronger after several weeks. Those to the St. Louis strain were also present in 3 of these sera but showed an initial potency, indicating that they had probably been acquired before the attack of encephalitis.

The antibodies appeared, perhaps weakly, as early as 8 days after the onset in those cases with a good recovery. With the sera examined from the groups shown in Table III, positive neutralization for the equine virus could be obtained at various periods up to one year after the onset. In 7 instances they were still present after 1 year or a little over, in 1 case at 2 years and in 2 others after 3 years and 2 months since recovery. There is every reason to believe, therefore, that the antiviral substances, if once established for this particular virus, may remain in the human serum for as long a time as for any other neurotropic virus disease. They were also found to

TABLE IV

*Positive Neutralization Tests According to Age Distribution*

| *County* | *Type of Virus* | *Total No. of Cases Tested* | *Under 5 Years* | *Total No. of Cases Tested* | *5 to 20 Years* | *Total No. of Cases Tested* | *Over 20 Years* |
|---|---|---|---|---|---|---|---|
| Fresno 1937–38 | Western Equine (Br strain) | 3 | 2 + (1 weakly) | 6 | 3 + | 14 | 7 + |
| | St. Louis encephalitis | | 1 + | | 2 + | | 7 + |
| | Both viruses | | 1 + | | 1 + | | 4 + |
| Kern 1938 | Western Equine (Br strain) | 7 | 4 + | 8 | 2 + of 7 tested | 11 | 3 + |
| | St. Louis encephalitis | | 1 + | | 6 + | | 6 + |
| | Both viruses | | 0 | | 2 + | | 4 + |
| Tulare 1937–38 | Western Equine (Br strain) | 1 | 0 | 12 | 1 + out | 9 | 4 + |
| | St. Louis encephalitis | | 1 + | | 6 + | | 7 + |
| | Both viruses | | 0 | | 1 + | | 2 + |

persist in the stored material. A few sera collected in August, 1937, were found to be positive upon testing in February, 1939, about a year and a half. There was no way of knowing, however, whether the antibody content had depreciated considerably, since no tests for the equine virus had previously been made. It was also noticed that the antiviral substances for the St. Louis virus persisted after about 2 years' storage. In several instances titrations were made on the same sera at different intervals, and a decline in titer was noticed.

Because of the presence of protective substances for the two different viruses in the blood of many of these encephalitic patients where clinical symptoms were similar, it is difficult to decide as to the etiological factor, especially where the actual virus had not been found. It seems very probable, however, that antiviral bodies for the St. Louis strain were present in many of the patients for some time. Many of the people belonged to the transient labor group and probably had acquired these antibodies in Missouri, Oklahoma, or Texas. If that were so, one would expect the greater number positive for the St. Louis strain to be found among the older age groups. Also one would expect the appearance of both types of antibodies in one serum among the latter group and the presence of one or the other among the younger people. This view seems justified as observed by reference to Table IV where the positive neutralization tests against the two viruses are tabulated for the counties of Kern, Tulare, and Fresno, according to the age distribution. As the age increases the number of positive tests to both strains also increases and there are more positives to one type of virus alone among the younger children than among the adults. There were, however, more encephalitis cases among the adults in these particular groups, 34 over 20 years of age, 26 from 5 to 20, and 11 under 5 years, with a predominance of the positive tests for the St. Louis virus (20 of the 34) among the oldest group. More positive neutralizations to the equine strain than to the St. Louis appeared among the younger people. One therefore may be justified in assuming that where a positive neutralization has been obtained for a particular virus alone among the children it is most probably the etiological agent of the neurotropic disturbance involved.

### EPIDEMIOLOGY

Since 1934, in California there has been noticed a change in the type of labor population inhabiting the two Central Valleys where the majority of the large agricultural industries are located. Where formerly the incoming laborers had been Mexicans, Filipinos, or southeastern Europeans, now they were migrants from Missouri, Oklahoma, or Texas, who were unable to cope with the financial depression and the dust storm problems at home and were now looking for the hoped-for land of opportunity. Coming in largely by the southern route they settled in the Valley regions from Kern County at the southerly end, working northward with the change of crops (Figure II). Most of them were poor, the living quarters most inadequate and sanitation far from favorable. In consequence whatever diseases occurred in the vicinity usually appeared among this group. Among the people coming to the Kern County Hospital to be bled for the neutralization test after recovery from a neurotropic disturbance, 14 out of 37 had some connection with Oklahoma, Missouri, or Texas. The whole subject of the migrant labor problem in California has been very graphically presented by a recent article in *Fortune*.[8] Residents from the middle and southwestern states have

gradually been moving toward the coast since 1930, with the greatest influx after 1934.

As noted, 1934 was the earliest date available when diseases of the central nervous system were reported in people whose sera were later shown to have neutralizing antibodies for the St. Louis virus. For the population from Kern County northward, all of these early cases were in the Central Valleys. From then on the number showing these antibodies seemed to increase and more frank cases of encephalitis were recorded for these areas. This was especially noticeable for the Tulare region where the sera obtained were more often positive to the St. Louis virus than to the equine (Table I). Did the migrants bring in the St. Louis strain and start an endemic area for this disease, which in many instances might be clinically difficult to distinguish from non-paralytic poliomyelitis? and then were certain other conditions superimposed that initiated an outbreak of yet a third type of central nervous system disturbances which could be particularly invasive for the rural population?

From certain epidemiological observations it seems likely that from Kern County northward some such factors have been at work during the past 4 or 5 years. Poliomyelitis was undoubtedly already endemic in many of the Valley towns. From 1934 to 1937 the

migration westward occurred in the largest proportions and with it most probably a few cases of the St. Louis type of encephalitis that may have helped immunize some of the population. In addition, a third neurotropic virus, that of equine encephalomyelitis, acquired significance for human welfare.

Previous to 1937 there had been a series of dry years throughout this part of the state, but during 1937 and 1938 the rainfall was unusual. The snow was heavy in the mountains, there were late rains with subsequent floods in the river areas and dry lakes were filled, tules grew where they had not been for years and much water was present in the central lands where the larger towns are found. As a result many mosquitoes appeared and were everywhere noticeable. Although no insect has as yet been actually implicated in the spread of the equine disease in the field, yet from the experimental work of Kelser,[9] Merrill and Ten Broeck,[10] Madsen, Knowlton, and Rowe [11] and Giltner and Shahan,[12] it seems most probable that a biting insect such as the mosquito is an important factor in transmission of this virus. They have all been able experimentally to produce the disease by means of several species of infected mosquitoes.

Whether or not a vector is responsible for the dissemination of the St. Louis virus is still an open question. Because of the negative results for mosquito transmission by Leake, Musson, and Chope in 1933,[13] the respiratory route of infection has been especially favored for this disease, largely because of the ease of intranasal transmission in mice. However, Webster, Clow, and Bauer [14] demonstrated later that *Anopheles quadrimaculatus* if infected can retain this virus for life, while Lumsden[15] has presented epidemiological evidence to show that *Culex pipiens* might have been implicated during the St. Louis epidemic in 1933. Subsequently Mitamura and Yamada, as reviewed by Inada,[16] have experimentally transmitted the virus of the Japanese B encephalitis by means of both *Culex pipiens* and *Culex tritaeniorhynchus*.

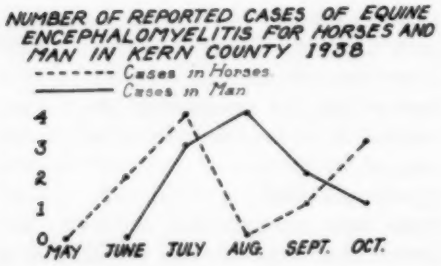
Although the disease of equine encephalomyelitis has been endemic among the horses in the San Joaquin and Sacramento Valley regions for many years, especially recognized since 1930 when the virus was first isolated by Meyer, Haring, and Howitt,[17] there has been no noticeable association with any similar form in man. It is true that 3 cases occurring in veterinarians or persons in close contact with horses had been reported in 1932 by Meyer,[18] but there was no associated outbreak among the general population. The largest epizoötic among horses had appeared from 1930 to 1932 and scattered cases were subsequently reported but not in epidemic proportions. For that reason after the first appearance of a definite human encephalitis around Fresno in 1937, no connection was made between the two forms. It now seems apparent, however, that the virus was the causative agent of many of the cases and probably of the fatal ones, especially as at least 3 of the patients in Fresno County and 2 in Kern who were ill in 1937 had antibodies for the equine type alone when tested in 1938. Since no virus was actually recovered from any of them in 1937, the true nature of the disease remained unrecognized until the following year.

From reports of the veterinarians in both Kern and Fresno Counties in 1938 there was no unusual number of sick horses but the mosquitoes were very prevalent. One might wonder, if the mosquitoes were the vectors in the equine outbreak in 1930, why human cases were not recorded and why they should now appear when the disease in

horses had subsided. One can only surmise that the unusual climatic conditions have brought the insects closer to the human population areas. As shown in the map the people with encephalitis having positive antibodies for the equine virus were found in the drainage regions, in the central portions of the valleys and mainly on the outskirts of the larger towns. Many cases of encephalitis were reported from the cities of Bakersfield and Fresno, but the individuals usually lived in streets bordering on the outskirts, near open fields where the wind could waft insects from the undrained areas. There is also the possibility that the unusual climatic conditions had encouraged the increase of some new vector common to both man and animals taken in combination with perhaps another animal reservoir than the horse.

By the courtesy of Henry Bye of the Kern County Health Department, Figure I was drawn from figures taken from their records for the human cases and from those of Dr. Edwards for the equine. A record is given of the monthly appearance during 1938 of human and of equine cases in the same localities. Apparently only 10 horses were treated in areas where human encephalitic cases were found, 8 of which were within a 4 mile radius of Bakersfield, and the others not so many miles away. The peak for the horses came in July, and a month later for man. The disease in the animals seemed to drop off and then re-occur later, with another rise in October. The number of equine cases was so small, however, that one cannot draw definite conclusions.

FIGURE I




Most of the human encephalitic cases considered to be of the equine type by reason of the neutralization test or the isolation of the virus, have come from the areas shown in the map. In Kern County 5 patients were within the 4 mile radius of Bakersfield or if in the town were on the outskirts, 2 were from Buttonwillow, 2 near Edison, while 6 cases of encephalitis were reported from the Arvin district with 2 of the equine type. In Fresno County there were 5 patients within a 5 mile radius of Fresno and the others from a group of closely associated small towns not far from Fresno, 3 from Sangar, 2 from Kerman, and 1 each from Selma and Del Rey. Several individuals were reported as having encephalitis at Fowler but no definite results were obtained. In Tulare the people were also from associated districts where mosquitoes were most prevalent.

It seems very probable that a number of the cases reported as encephalitis in the Valley counties northward were also of the equine type, but unfortunately material was not available from them again and most of the sera first collected were exhausted. However, sufficient evidence was obtained from the few sera on hand to indicate a more general distribution of the equine virus in man throughout both of the valley regions than may have been suspected. Since the epizoötic of 1930 and 1931 was widespread throughout both the Sacramento and San Joaquin Valleys (morbidity at that time being about 6,000 horses with an average mortality of 47 per cent, according to Meyer [18]), it is not unreasonable to suppose that the human form had been present, now that proof has been given of the infectiousness of this virus for man. Two

of the 3 human cases reported by Meyer in 1932 [18] came from the Sacramento district and the third from Madera above Fresno. Serum was obtained again from the latter person 6 years after recovery but gave weakly positive protection for the Br strain.

Since nearly all of the positive cases in the counties of the lower San Joaquin Valley had possibilities of exposure to some form of vector but not necessarily to sick horses, especially in regard to the infants, it seems very likely that a biting insect has been the responsible agent for the recent outbreak of the equine disease.

## DISCUSSION

During 1938 the actual recovery of the equine virus from fatal cases of human encephalitis was reported first by Fothergill, Dingle, Farber, and Connerly [19] and Webster and Wright [20] for the eastern form in Massachusetts, and then by Howitt [6] for the western in California. A more detailed account of the eastern outbreak was then given by Feemster [21] and also by Wesselhoeft, Smith, and Branch [22] who reported the histories of 8 fatalities in Massachusetts with recovery of the virus from 5. At about the same time Eklund and Blumstein [23] in Minnesota presented a clinical account of cases of human encephalitis closely associated with an equine epidemic during 1937. Since neutralizing antibodies for the western equine virus were found in the sera of 3 recovered patients, this strain was considered to be the etiological agent.

Prior to these reports the equine disease had not been incriminated for man except for the 3 cases already described by Meyer in 1932. It seems highly probable, however, that many acute cases of encephalitis without recovery of an active agent could have been attributed to this disease and that both non-paralytic poliomyelitis and the St. Louis type of encephalitis were confusing factors in many instances. It was interesting that the latter virus was evidently intermixed in California in such a way as to cause confusion in differentiation. Since the actual virus has not as yet been recovered from these areas, it is still uncertain whether or not true cases have been present. It has been shown by Wooley and Armstrong [3] and by Muckenfuss, Smadel, and Moore [5] that antibodies to this virus may be found among the general population in an endemic area, comparable to what has been reported for poliomyelitis. For this reason, as demonstrated by Smith and Moore,[24] too much diagnostic significance should not be placed on their presence in acute cases unless the serum of the patient is negative during the early stages of the disease and later shows a rise in antibody content after recovery. So far in the California areas affected there has not been any generalized immunization to the equine virus, because only those persons showing an acute disturbance of the central nervous system developed antiviral substances. Normal people and those having acquired poliomyelitis in the Bay Region were all negative, with the exception of one individual in the San Joaquin Valley whose history was unknown.

This differentiation in the presence of antibodies to both viruses leads one to suspect that the equine form has rather recently become of human significance, probably because of the sudden appearance of a new vector or perhaps a new reservoir host. The eastern virus has lately been recovered in nature from both the pigeon [25] and the pheasant [26] so that some such host may also be present in the West. Whatever is later proved, it seems warrantable to assume that at present the occurrence of protective substances for the western equine virus has some diagnostic value in the Californian regions affected and that this disease

first became of marked significance to human welfare during 1937. Sporadic cases very likely occurred throughout the northern and central valleys during the previous years but were reported either as non-paralytic poliomyelitis or as some form of encephalitis of unknown etiology. Sera were positive for 2 cases having their onset in 1935 and for 1 in 1936. A more complete survey of the population would undoubtedly show a larger number of positive tests. On the other hand, the simultaneous presence in a serum of antibodies for the St. Louis virus may not have as much value because of a more generalized immunization in certain Valley areas.

## SUMMARY

1. An outbreak of the western type of equine encephalomyelitis occurred among the human population of the two large Central Valleys in California during 1937 and 1938.

2. The western strain of the equine virus was recovered from the brain and the blood, respectively, of 2 human cases of encephalitis in this region. Both strains were shown to be immunologically and serologically similar to the virus of equine origin.

3. Total number of cases of encephalitis tested throughout northern and central California:

Positive neutralization to the St. Louis virus: 49 (47.5 per cent) out of 103 sera.

Positive neutralization to the equine virus (Br): 32 (37.0 per cent) out of 86 sera.

4. Number of cases of encephalitis tested in Fresno, Tulare and Kern Counties only:

Positive neutralization to the St. Louis virus: 37 (52.1 per cent) out of 71 sera.

Positive neutralization to the equine virus (Br): 26 (44 per cent) out of 59 sera.

5. Positive neutralization to both viruses: 18 (26 per cent) out of 69 sera.

6. Of a total of 63 apparently normal individuals, contacts and non-contacts, residing in both the Bay Region and the Valley districts, 8 had neutralizing substances for the St. Louis virus and 1 for the equine. With the exception of 1 for the St. Louis type, all of the positive group resided in the San Joaquin Valley.

7. Of a total of 47 individuals tested for equine virus and 60 tested for the St. Louis strain among groups diagnosed as having poliomyelitis in both the Bay Region and in the Valleys, the only positive sera for either virus were from those in the San Joaquin Valley.

8. Fourteen of 21 sera positive to the Br equine strain also gave strongly positive neutralization with the western virus of equine origin (Californian), while 4 were weakly positive.

9. Neutralizing antibodies for the Br equine virus could be obtained as early as 8 days after onset of the disease and were found in some individuals after 1, 2, or even 3 years since recovery. Positive sera for both the St. Louis and the equine strains retained their neutralizing power after storage for from 1 to 2 years, but potency was decreased.

10. In the encephalitic series as the age increased, the number of positive neutralization tests to both strains increased and there were more positives to a single type of virus alone among the younger groups than among the older. The predominance of positive tests for the St. Louis virus was among the older people, while that for the equine was among the younger.

11. No very definite relationship was noted between cases of the equine disease in horses and those in man, since the former did not reach epidemic proportions of those showing clinical symptoms and the observed cases were only occasionally in concurrent areas. Swampy lands and many mosquitoes, however, were in close association with the districts affected.

12. From the epidemiological evidence it seems likely that the equine virus was transmitted to man through some vector.

13. The St. Louis strain was brought in because of the migrant labor population.

## CONCLUSION

The western virus of equine encephalomyelitis has been found capable of infecting man in certain districts of northern and central California and neutralizing substances for this strain have been found in the sera of 32 (37.0 per cent) out of 86 encephalitic cases diagnosed in 1937 and 1938. Forty-nine (47.5 per cent) out of 103 sera of this group gave positive protection tests for the virus of St. Louis encephalitis.

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NOTE: Appreciation is extended to the Health Departments of Kern, Fresno, and Tulare Counties, respectively, for their kind coöperation in obtaining material and in extending hospital facilities for the collection of sera.

# Detection of Trichinella Infestation in Hogs by the Intradermal Test*

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THE work of recent years has shown that in the United States human infestation with *Trichinella spiralis* is much more common than was heretofore believed. Thus, 11 investigators [1-11] whose papers have been published since 1930, report a total of 5,941 human cadavers examined, of which 935 were infested with trichinella, an incidence of 15.7 per cent. The control of trichinella infestation is therefore a subject which should interest the health officer.

It has long been recognized that the routine microscopic examinations of pork flesh, such as are carried on in various European countries, are quite unreliable, failing in many instances to reveal an existing infestation. Bachman,[12] in 1928, called attention to a useful precipitin test, and in a later article in the same year described a clearly defined, delayed type of skin reaction in rabbits and guinea pigs artificially infected with trichinella. Some time afterward, Augustine and Theiler [13] began a well planned study of both the precipitin and the skin tests, applying them not only to swine but also to humans. In general, they followed Bachman's technic for preparing the antigen for the intradermal test. They also emphasized the immediate character of the skin reaction obtained.

The present study was made to determine the value and accuracy of the intradermal test in detecting trichinous animals in a group of city-owned, garbage-fed hogs.

## ANTIGEN PREPARATION

Albino rats weighing between 100 and 125 grams were starved 24 hours and then fed about 5 gm. of cheese which contained approximately 100 trichina larvae per gm. After 6 weeks, the animals were killed, skinned, and eviscerated. The carcasses were ground and then set aside for digestion in a fluid made by dissolving 5 gm. of pepsin in a liter of warm tap water and adding 7 cc. of concentrated hydrochloric acid. For every 50 gm. of meat, 2 l. of digestive fluid were used.

The apparatus and method for the digestion were similar to those used by Bozicevich.[14] A 4 l. funnel, to the stem of which a centrifuge tube was attached by a short piece of rubber tubing, was used. A pinchcock was placed at the end of this tubing to permit closing, and the centrifuge tube containing larvae was removed after digestion of the meat. A crossbar, made by tying together two pieces of glass tubing, was placed in the funnel, and over this were laid 4 layers of cheesecloth. The digestive fluid was poured into the funnel and the meat carefully placed on top of the cheese-

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cloth. The funnel was put in an incubator at 38° C. for 18 hours, at the end of which, the trichinae which had been liberated from their cysts by the digestive fluid had gravitated to the bottom of the centrifuge tube. The pinchcock on the rubber tubing was then closed and the centrifuge tube removed. The supernatant fluid along with the layer of sediment was removed by means of a pipette.

To remove some of the débris which had settled with the larvae at the bottom of the centrifuge tube, a 250 cc. funnel was set up in a manner similar to the one described above, the only difference being that no crossbar was used to support the cheesecloth. About 200 cc. of warm tap water was poured into the funnel and into this the contents of the centrifuge tube containing the trichina larvae mixed with some débris was washed.

In about 30 minutes, all of the larvae almost free of visible contamination had passed through the gauze and settled to the bottom. The rubber tubing was closed with the pinchcock and the centrifuge tube removed. The supernatant fluid was removed by a pipette and the larvae were washed several times with 0.5 per cent sodium bicarbonate solution. After each washing, the tube was set aside for 30 minutes, during which the larvae settled to the bottom and the supernatant fluid was removed.

At the third washing, a biuret test was performed on the supernatant fluid and found to be negative. Therefore, no further washings were done and the larvae were placed in an agate mortar and put into a vacuum desiccator containing phosphorous pentoxide. After evacuation of the air, the desiccator was placed in the refrigerator for 24 hours. The trichinae were then pulverized, weighed, and the powder extracted with 0.85 per cent sodium chloride solution, using 1:500 dilution. After extraction for 2 hours at room temperature, the suspension was placed in the ice box for 3 days, at the end of which time the suspension was placed in a water bath at 58° F. for 1 hour on 3 successive days.

A portion of this suspension was then centrifuged at 3,000 revolutions per minute for 20 minutes. The supernatant fluid constituted the antigen used in our test. Prior to injecting the pigs, the antigen was standardized with the aid of artificially infested rats. The rats were divided into two groups of 12 each. The first group was injected with 0.1 cc. of the uncentrifuged antigen, using 3 rats each for dilutions of 1:500, 1:2,500, 1:5,000, and 1:10,000. This was repeated on the second group of 12 rats with centrifuged antigen. With the exception of the 1:500 concentration, Coca's solution (sodium chloride 0.7 per cent, sodium bicarbonate 0.05 per cent, phenol 0.4 per cent) served as the diluent and was also used for the control injection. Physiological saline was used in the control injection for the rats which received the 1:500 dilution of antigen. In all 24 rats, each dilution gave immediate type positive reactions, with wheals which at the end of 20 minutes averaged about 2.0 cm. in diameter. This indicates that the active principles are apparently in the supernatant eluted portion. It was this portion that was used for the intradermal skin test of hogs.

### TESTING THE PIGS

The great majority of pigs used were of the graded Chester White and graded Yorkshire White breeds with several animals of the Poland China variety. The hogs were fed with uncooked swill. The garbage often contained considerable quantities of frankfurters, smoked sausages, bologna-style sausages, smoked ham, and other pork products. On no occasion was the garbage sterilized

TABLE I

| *Pig No.* | *Skin Reaction* | *Diaphragm Digestion Findings* | *Observations* |
|---|---|---|---|
| 19 | Negative | Negative | Papillary dermatitis |
| 20, 24, 31 | Positive | Positive † | Normal |
| 23, 34 | Positive | Positive * | Normal |
| 46 | Positive | Positive ‡ | Normal |
| 48 | Positive | Positive † | Normal |
| 4A | Positive | Negative | Wide area of erythema |
| 5A, 6A, 15A | Positive | Positive * | Normal |
| 7A | Positive | Negative | Papillary dermatitis of the skin |
| 26A | Negative | Positive (very light infestation) | Heavy thickening of the epidermis |
| 35A | Negative | Negative | Scaly dermatitis |
| NOTE: 44 pigs | Negative | Negative | Normal |

* Light Infestation (1 to 10 larvae per gm.)
† Moderate Infestation (11 to 25 larvae per gm.)
‡ Heavy Infestation (26 or more larvae per gm.)

before being fed to the swine. The pigs averaged about 1 year of age before slaughter and in almost all cases, were killed 1 to 5 days after they were injected. The skin test was performed on the inner surface of the distal tibial region. The hair was removed with a clipper from a 2½ sq. in. area, the surface washed with alcohol, and 0.1 cc. of the antigen was injected. The control injection of Coca's solution was given approximately 4 cm. from the site of the injected antigen.

In Tables I and II, we list only those pigs which showed deviations from the normal. The antigen used in the series shown in Table I was diluted with Coca's solution to a 1:10,000 dilution. Readings of the injected site were usually made in from 20 to 45 minutes. The wheal which forms is 1.5 to 2.5 cm. in diameter, and assumes a faint purplish-red hue with definite margins, without any noticeable erythema or edema of the surrounding tissues. Usually at the time of the reading, the bleb first formed by the control injection had almost disappeared.

In order to determine whether the skin diagnosis was correct, the diaphragms of the hogs were removed at the time of slaughter and digested as described in the preparation of the antigen, after removal of fat and grinding. For every 50 gm. of meat, 1 l. of digestive fluid was used. A convenient modification was the use of percolators of approximately 4 l. capacity instead of funnels in the digestion of the meat. The percolators were kept at 37° C. for 18 hours, at the end of which the attached centrifuge tubes were removed and examined for trichina larvae.

In the series shown in Table II, the antigen was diluted with 0.85 per cent saline containing 0.5 per cent phenol instead of Coca's solution, and this saline was used for the control injection.

Pigs 4A, 7A, 55, and 84, which gave positive reactions, showed no trichina larvae in the diaphragms. In pig 4A, there was an erythema of the entire clipped surface with a slight elevation 1 cm. in diameter. In pigs 7A, 55 and 84, there was a papillary dermatitis of the skin which, at the time of reading, showed a widespread erythematous area with several papillae. Pig 26A, which gave a negative skin test, showed 250 larvae in the diaphragm that

TABLE II

| Pig No. | Skin Reaction | Diaphragm Digestion Findings | Observations |
|---|---|---|---|
| 10, 12 | Positive | Positive ‡ | Normal |
| 47A | Positive | Positive * | Normal |
| 55 | Positive | Negative | Papillary dermatitis |
| 58 | Negative | Negative | Marked papillary dermatitis and thickening of the epithelium |
| 61 | Negative | Negative | Papillary dermatitis |
| 62 | Negative | Negative | Marked papillary dermatitis |
| 64 | Negative | Negative | Tumor, midline between umbilicus and groin |
| 74 | Positive | Positive † | Normal |
| 84 | Positive | Negative | Marked papillary dermatitis |

NOTE: The clipped skin of animals 85 to 179, or 83 pigs, received no alcohol washing prior to injection.

| | | | |
|---|---|---|---|
| 99 | Negative | Negative | Mild dermatitis, thickening of the epithelium |
| 100, 124 | Negative | Negative | Papillary dermatitis |
| 107, 109, 111 | Positive | Positive * | Normal |
| 125, 134, 136 | Positive | Positive * | Normal |
| 135, 140, 145 | Negative | Negative | Papillary dermatitis |
| 148, 149, 154 | Negative | Negative | Papillary dermatitis |
| 155 | Negative | Negative | Erythema of legs and abdomen |
| 167, 168, 170 | Negative | Negative | Papillary dermatitis |
| 173, 175, 179 | Negative | Negative | Papillary dermatitis |
| NOTE: 119 pigs | Negative | Negative | Normal |

* Light Infestation (1 to 10 larvae per gm.)
† Moderate Infestation (11 to 25 larvae per gm.)
‡ Heavy Infestation (26 or more larvae per gm.)

weighed 365 gm., or a very slight infestation. This pig had a marked epidermal thickening which was difficult to inject.

In pigs 85 to 179, no alcohol was used to wash the skin surface prior to the injection.

## DISCUSSION

Altogether, 211 pigs were intradermally injected with trichinella antigen and skin readings were made within 20 to 45 minutes, the average being 30 minutes. Of these 25 were diagnosed as positive and 186 as negative. Upon corroborative examination of the digested diaphragms, it was found that 21 out of 25 diagnosed as positive were trichinous, and 185 of the 186 diagnosed as negative were non-trichinous. Thus, 206 out of 211 pigs were correctly diagnosed, an accuracy of about 97 per cent. In this series, the incidence of trichinosis was 10.4 per cent.

It is advisable not to wash the skin surface with alcohol before injection of the antigen. This is especially important when a papillary dermatitis exists,

because of possible distension and erythema.

In pigs 85 to 179, where no alcohol was used, there were 16 pigs with papillary dermatitis, and in all the correct diagnosis was made. It is suggested, therefore, that pigs 7A, 55, and 84, with papillary dermatitis, may have been incorrectly diagnosed for this reason.

One pig, 26A, passed by the skin test as not trichinous, was found at autopsy to be infected. This pig had marked epidermal thickening, and was difficult to inject. In such cases, there is a possibility that the needle does not come into contact with the capillary circulation. Under these circumstances, no reaction will result even if the animal is trichinous. Pig 26A showed 250 larvae in a diaphragm weighing 365 grams. This is a very light infestation. It is important to remember that the intensity of the skin reaction bears no relation to the degree of infestation. In pig 111 the skin test gave a wheal, 2½ cm. in diameter and well elevated in 25 minutes. From the digested diaphragm, only 4 larvae per gm. were obtained, denoting a light infestation. In this series of cases the small error in the skin tests was in the direction of safety, the test being more likely, now and then, to diagnose a normal pig as trichinous, than to permit a trichinous pig to pass as normal.

## SUMMARY

Two hundred and eleven pigs were skin-tested with trichinella antigen. All the pigs had been fed on uncooked garbage. Post-mortem examination of the digested diaphragms was made as a check on the diagnostic accuracy of the skin test. In 4 out of 25 pigs diagnosed as trichinous by skin tests, no evidence of infestation was found at autopsy; in 1 pig out of 186 diagnosed as non-trichinous, post-mortem examination showed trichinosis. Of 211 pigs skin-tested, 206 were correctly diagnosed as to the presence or absence of trichinous infestation. This is an error of less than 3 per cent. The error, moreover, is in the direction of safety, being more likely now and then to diagnose a normal animal as trichinous, than to pass a trichinous pig as normal.

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# Experimental Infection of Dermacentor Andersoni Stiles with the Virus of Lymphocytic Choriomeningitis*

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ALTHOUGH only 4 years have elapsed since Armstrong and Lillie [1] showed that lymphocytic choriomeningitis is an infectious disease caused by a virus, the evidence indicates that the etiologic agent can be transmitted in several ways. Traub [2] has shown that the virus is discharged in the urine and nasal secretions of infected mice. Findlay and Stern [3] succeeded in infecting 3 of 20 mice by nasal instillation of the virus. Finally Wooley, Armstrong, and Onstott [4] have been able to transmit infection occasionally by instilling the virus into the urethra or vagina of monkeys. They have also demonstrated the virus in the seminal fluid and testicular tissue of infected animals. On the basis of these findings, together with the fact that antibodies against the virus were found by them more commonly in adults and especially in criminal and merchant seamen classes, these investigators have suggested a venereal route of transmission.

However, the higher incidence of antibodies against this virus in criminals and merchant seamen may be accounted for by the greater opportunity for contact with infected mice and rats. Thus Findlay, Alcock, and Stern [5] have isolated the virus of this disease from a patient who lived in an old house overrun with rats and mice. These authors intimate that they believe this may be a general source of infection.

Naturally infected mice have been found by Traub [2] and Lepine and Sautter.[6] It is possible that other species of rodents such as ground squirrels and rats may also be naturally infected with lymphocytic choriomeningitis. Traub [7] and Rivers and Scott [8] noted that normal mice and guinea pigs are not readily infected by contact with infected animals, an observation which we have confirmed. The manner in which naturally infected mice contracted the disease is unknown. These findings suggested the possibility that blood sucking arthropods may be one means of transmitting the virus of this disease from rodent to rodent.

In the experiments reported here the

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Rocky Mountain wood tick (*Dermacentor andersoni,* Stiles) was used as a convenient test vector. These were laboratory reared ticks obtained through the courtesy of Dr. R. R. Parker of the Rocky Mountain Laboratory of the U. S. Public Health Service and believed by him to be free from other infectious diseases. We noted no evidences of carriage of other diseases by the ticks during the course of our work, and control studies with uninfected ticks were always negative.

The strains of mice and guinea pigs used in our experiment were shown not to be naturally infected with lymphocytic choriomeningitis by subjecting them to intracerebral injection of starch, broth, or normal brain emulsion, procedures which have been found will activate latent infection with this virus into a typical syndrome.[5] Our experimental animals were also tested for immunity resulting from possible previous subclinical infections by injecting large numbers intracerebrally with virus. No evidence of immunity was discovered.

Adult forms of the tick were fed by confining them on the shaved bellies of guinea pigs under screw-top screen capsules such as described by Jellison and Philip.[9] Since larvae and nymphs often become stuck to the adhesive tape girdle, it was necessary to use the technic described by Wolbach[10] for feeding the immature stages. These forms were introduced into a covered jar containing the guinea pig on which they were to feed. The inner surface of the jar near the top was coated with vaseline to keep them from escaping. At the end of three days the guinea pig with attached ticks was transferred to a similar jar and the first jar steamed to kill unattached ticks. The animal remained in the second jar until the engorged nymphs or larvae had dropped. The latter were then collected and transferred to test tubes for further studies.

Rivers and Scott[8] stated that the blood of guinea pigs inoculated intracerebrally is infectious for mice as early as the 4th day after inoculation. We have been able in three experiments to show that, following combined intracerebral and subcutaneous inoculation

TABLE I

*Persistence of the Virus of Lymphocytic Choriomeningitis in the Blood Stream of Infected Guinea Pig 3140 * (First Experiment)*

| *Guinea Pig No.* | *Day Following Inoculation* | *No. of cc. of Heart's Blood Inoculated †* | *Result* |
|---|---|---|---|
| 3439 | 1 | 0.2 | Died in 9 days |
| 3402 | 2 | 0.2 | Died in 9 days |
| 3405 | 3 | 0.2 | Died in 10 days |
| 3406 | 4 | 0.2 | Died in 9 days |
| 3407 | 5 | 0.2 | Died in 8 days |
| 3408 | 6 | 0.2 | Died in 10 days |
| 3409 | 7 | 0.2 | Killed accidentally on 5th day ‡ |
| 3413 | 8 | 0.2 | Died in 8 days |
| 3145 | Control ** | 0.2 | Negative |

* Guinea pig 3140 died on the 8th day following intracerebral (0.2 cc.) and subcutaneous (2 cc.) inoculation with 10 per cent guinea pig brain emulsion infected with virus strain W. E. 9210.

† Heart's blood in 2 per cent potassium oxalate was injected intracerebrally.

‡ This guinea pig had a typical temperature rise and lost 50 grams of weight before being accidentally strangled on the 5th day following inoculation.

** The control guinea pig was inoculated with heart's blood drawn just before injecting guinea pig 3140 with the virus. Later this control guinea pig was found to be susceptible to subcutaneous inoculation (0.2 cc. of 1/100 dil.) of infected guinea pig brain emulsion.

of the W. E. 9,210 strain of virus, active virus appears in the blood stream of infected guinea pigs as early as 24 hours after inoculation and persists until the death of these animals. Sample protocols are outlined in Tables I and II. Virus in sufficient quantities to infect mice appeared in the blood stream of guinea pigs 48 hours after rubbing virus on their lightly scarified skins. The scarification was not deep enough to draw blood. From these studies it appeared that feeding ticks might become infected at any time from one day after infection until death.

Preliminary experiments in which ticks were collected after feeding on infected animals, ground up, and injected into normal animals indicated that larvae, nymphs, and adults, both male and female, can be experimentally infected in this manner. Infection persisted in adult ticks for at least as long as 13 days. Stage to stage transmission of the virus in ticks was also demonstrated by injecting ticks which had engorged in their previous stages on infected guinea pigs. The eggs of adult females which had engorged on infected guinea pigs also

TABLE II

*Persistence of the Virus of Lymphocytic Choriomeningitis in the Blood Stream of Infected Guinea Pig 3436* (Third Experiment)*

| *Day Following Inoculation* | *Cage No.* | *Virus Dilution †* | *Day of Death of Mice* | *No of Mice Surviving* |
|---|---|---|---|---|
| 1 | 75 | 1/10 | 9, 11..... | 0 |
| | 76 | 1/100 | 11..... | 1 |
| | 77 | 1/1,000 | ..... | 2 |
| 2 | 78 | 1/10 | 8, 10..... | 0 |
| | 79 | 1/100 | 8, 8..... | 0 |
| | 80 | 1/1,000 | 9..... | 1 (1) ‡ |
| 3 | 82 | 1/10 | 7, 8..... | 0 |
| | 83 | 1/100 | 8, 9..... | 0 |
| | 84 | 1/1,000 | 9, 9..... | 0 |
| 4 | 85 | 1/10 | 9, 9..... | 0 |
| | 86 | 1/100 | 14, 14.... | 0 |
| | 87 | 1/1,000 | 8, 8..... | 0 |
| 6 | 88 | 1/10 | 9, 10..... | 0 |
| | 89 | 1/100 | 8, 10..... | 0 |
| | 90 | 1/1,000 | 11..... | 1 (1) ‡ |
| 6 | 92 | 1/10 | 9, 10..... | 0 |
| | 93 | 1/100 | 10, 10.... | 0 |
| | 94 | 1/1,000 | 10..... | 1 (1) ‡ |
| 7 | 95 | 1/10 | 7, 9..... | 0 |
| | 96 | 1/100 | 9, 11..... | 0 |
| | 97 | 1/1,000 | 9, 10..... | 0 |
| 8 | 98 | 1/10 | 6, 8..... | 0 |
| | 99 | 1/100 | 7, 8..... | 0 |
| | 100 | 1/1,000 | 9, 12..... | 0 |
| Control ** | 74 | 1/10 | ..... | 2 |

* Guinea pig 3436 died on the 8th day following intracerebral (0.2 cc.) and subcutaneous (2 cc.) inoculation with 10 per cent guinea pig brain emulsion infected with virus strain W. E. 9210.

† 0.03 cc. of heart's blood in 1 per cent sodium citrate solution diluted in buffered saline was injected intracerebrally into two mice for each dilution.

‡ Indicates number of surviving mice that developed typical signs of lymphocytic choriomeningitis but recovered.

** The control mice were inoculated with heart's blood drawn just before injecting guinea pig 3436 with the virus.

contained sufficient virus to kill guinea pigs injected with them. In one experiment the virus was shown to persist from infected adult ticks through their eggs and to the larval stage. It is of interest that the ticks which had engorged in their previous stage on an infected animal were apparently not infectious until they had engorged on a normal guinea pig. This parallels the phenomenon which occurs in Rocky Mountain spotted fever.

Although adult ticks probably would not migrate under natural conditions from animal to animal after they had once begun to feed, transmission experiments were carried on with them as well as with larvae and nymphs. In order to rule out the possibility of an extrinsic incubation period of the virus in the ticks, varying periods of time were allowed to elapse before transferring ticks from infected guinea pigs to normal animals as shown in Table III. All of the guinea pigs upon which the infected ticks were fed continued to gain weight in a normal manner, and none of them showed any significant temperature rise or other symptoms of the disease during the 28 days that they were observed. Three weeks after the infected ticks were removed, the guinea pigs were tested for immunity by subcutaneous inoculation of virus to which all succumbed with typical signs of lymphocytic choriomeningitis.

Similarly all attempts to transmit the virus by feeding adults which had engorged in their nymphal stage failed. In our earlier experiments we encountered failure in attempting to transmit the virus by feeding nymphs which had engorged in their larval stage on infected animals. Since these findings were first reported, however, experiments have been completed showing that nymphs, which had engorged in their larval stages on an infected guinea pig, successfully transmitted the virus of lymphocytic choriomeningitis to normal guinea pigs by feeding on them. It is probable that our earlier failures to transmit infection in this manner were due to attachment of an inadequate number of infected nymphs to the test animal. In a personal communication Dr. Parker states that some batches of ticks fail to feed well.

TABLE III

*Attempts to Transmit the Virus of Lymphocytic Choriomeningitis by Feeding Infected Adult Ticks on Normal Guinea Pigs*

| Tick Lot No. | No. of Ticks Fed | | Days Fed on Infected Guinea Pig | Days Elapsed Before Feeding on Normal Guinea Pig | Days Fed on Normal Guinea Pig | Result |
|---|---|---|---|---|---|---|
| | Male | Female | | | | |
| 3141 | 2 | 2 | 7 | ½ | 4 | Negative |
| 3410 | 8 | 3 | 4 | ½ | 6 | Negative |
| 3418 | 1 | 15 | 5 | ½ | 5 | Negative |
| 3110 * | 1 | 2 | 5 | 8 | 5 | Negative |
| 3469 | 2 | 4 | 5 | 14 | 5 | Negative |
| 3456 | 0 | 4 | 5 | 21 | 6 | Negative |
| 3111 (Control) | 2 | 5 | .. | .. | 10 | Negative |
| 3142 † (Control) | 2 | 4 | .. | .. | 10 | Negative |
| 3417 (Control) | 10 | 4 | .. | .. | 11 | Negative |

* Six infected nymphs were included in this lot.
† Seven unfed nymphs were included in this lot.

Another possibility of natural transmission remained. In relapsing fever and plague it is believed that infection is acquired mainly by rubbing the infected dejecta of the lice or fleas into the wound by scratching the bitten area. Since we had already demonstrated that virus could penetrate the lightly scarified skin, this possible route of transmission was investigated. Feces from feeding adult ticks which had engorged in their previous stage were found to be infective when applied to a small area (about 3 x 3 sq. cm.) of skin scarified too lightly to draw blood. It was also found that crushed adult ticks applied to scarified skin caused typical symptoms of the disease in guinea pigs. Control experiments with normal tick feces and crushed ticks were uniformly negative.

SAMPLE PROTOCOL

*Application of Infected Adult Tick Feces to the Lightly Scarified Skin of Normal Guinea Pig:*

I. Control—

April 8, 1939, sufficient feces were collected from 8 adult female and 6 adult male ticks engorging on infected guinea pig 4006, 8 days after the ticks had attached, to prepare a 10 per cent suspension in buffered saline. Guinea pig 4012 was inoculated subcutaneously (1 cc.) with infected tick feces suspension. *Micrococcus epidermidis* and hay bacilli were isolated from the infected tick feces suspension.

Temperature record of guinea pig 4012: April 8, 101.7 (normal temperature before injecting tick feces); April 9, 101.6; April 10, 104.2; April 11, 104.8; April 12, 103.9; April 13, 101.5 (labored breathing); April 14, animal found dead. This animal lost 32 grams in 7 days.

At autopsy the brain was very hyperemic, and the other organs were apparently negative. No bacteria were isolated from the brain tissue. A 10 per cent brain suspension was then prepared and injected subcutaneously (2 cc.) into guinea pig 4015 and intracerebrally (0.03 cc.) into each of 2 white mice (cage 1A). Both of the mice developed typical signs of lymphocytic choriomeningitis in 8 days but recovered. Twenty-seven days after the first inoculation both mice and 2 controls were inoculated intracerebrally (0.03 cc.) with a 10 per cent suspension of infected guinea pig brain. The mice were immune to the virus because they remained normal during the 30 days that they were observed, while the 2 control mice succumbed in 7 and 10 days respectively. Guinea pig 4015 developed typical signs of lymphocytic choriomeningitis and succumbed in 9 days. No bacteria were isolated from the brain tissue of guinea pig 4015.

II. Tick feces applied to the lightly scarified skin—

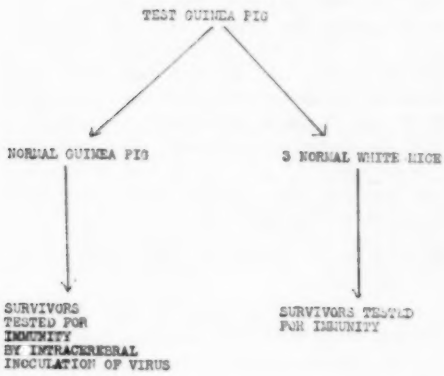
April 8, 1939, the same 10 per cent suspension of infected tick feces was also applied to the lightly scarified skin of guinea pig 4014 with a cotton swab. Scarification was accomplished by making 8 scratches in the shaven skin not deep enough to draw blood by means of a razor.

Temperature record of guinea pig 4014: April 8, 102.5 (normal temperature before applying tick feces); April 9, 102 (scratches not visible); April 10, 102.2; April 11, 104.8; April 12, 104.2; April 13, 105.5; April 14, 104.5; April 15, 106.6; April 16, 104.7; April 17, 103.2; April 18, 101.9; April 19, 98.1 (labored breathing); April 20, animal found dead. In 11 days this animal lost 68 grams.

At autopsy the brain was inflamed, and there was a diffuse bronchopneumonia in the lungs. Other organs were negative. No bacteria were isolated from the brain tissue. A 10 per cent brain suspension was then prepared and injected subcutaneously (2 cc) into guinea pig 4017 and intracerebrally (0.03 cc.) into each of 2 mice (cage 3). Both of the mice developed typical signs of lymphocytic choriomeningitis and succumbed in 7 and 9 days respectively. Guinea pig 4017 also developed typical signs of lymphocytic choriomeningitis and succumbed in 12 days. No bacteria were isolated from the brain tissue of guinea pig 4017.

In the successful transmission experiments typical symptoms were produced in all test animals and all animals that died showed typical pathology. Surviving animals in all experiments were shown to be immune to inoculation of the virus of lymphocytic choriomeningitis. As a final proof, the virus was shown to be present in the test animals by inoculation of a guinea pig and several white mice as shown in Figure I. In all experiments bacteriologic studies, conducted aerobically and an-

FIGURE 1


TEST GUINEA PIG
NORMAL GUINEA PIG
3 NORMAL WHITE MICE
SURVIVORS TESTED FOR IMMUNITY BY INTRACEREBRAL INOCULATION OF VIRUS
SURVIVORS TESTED FOR IMMUNITY

aerobically, indicated that only chance contaminants were present occasionally. Inoculation of animals with cultures of these organisms proved them to be non-pathogenic.

## CONCLUSIONS

Experimental infection of all stages in the life cycle of the Rocky Mountain wood tick with the virus of lymphocytic choriomeningitis has been demonstrated. Stage to stage transmission of the virus from larvae to nymphs, nymphs to adults, and generation to generation transmission from adults to eggs and larvae has also been shown to occur. Attempts to transmit the infection by feeding adults on normal animals after previously feeding them in either their nymphal or adult stages on infected guinea pigs were uniformly negative. Infection has been transmitted, however, by feeding nymphs which had engorged in their larval stage on an infected guinea pig. Infection has also been transmitted by applying infected crushed ticks or feces from ticks which had engorged in their previous stage upon infected animals to the scarified skin of guinea pigs.

It is suggested that other blood-sucking arthropods such as culicine mosquitoes, stable flies, and body lice may experimentally transmit the virus of lymphocytic choriomeningitis from rodent to rodent and possibly to man. If infection is transmitted from rodent to man, it is possible that this may occur by means of infected secretions or excretions such as may take place in fectious jaundice rather than by the bite of an arthropod.

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# The Physician's and the Nurse's Part in Health Education*

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HEALTH education has been claimed to be one of the major functions of each professional group engaged in the school health program. The physicians contend that they should do health education because they know the subject matter; the teachers claim that health education is their function because they know the method; the nurses maintain that they are the logical ones to teach health because, in addition to knowing sufficient subject matter and to having acquired some technics of presentation, they know their families and are in intimate contact with them. Implied in all these contentions is the concept that health education is primarily a matter of instructing parents and children on health subjects. The idea seems to be that if the proper professional worker gives instruction to those needing it, desirable results are assured. But the bare statement of the spoken or written word is not the sole means of bringing about the changes in attitude and behavior which we are trying to effect through our health education. *How* a statement is spoken or written may have far more significance for the person addressed than actually *what* is said. Actions, too, often confound the words accompanying the action so that the individual may learn something altogether different from what was intended.

So instead of discussing the controversial question of whether health teaching should be done by the physician, by the nurse, or by the teacher, we have chosen to focus attention on the educational possibilities that are inherent in many of the administrative procedures and policies that are part and parcel of our school health programs.

As a background for this discussion it might be well to recall some of the factors with which all of us are familiar that influence *what* and *how* people learn. First, one seldom learns just one thing from even a single experience. Ordinarily a number of things are learned from the same situation. A child not only learns that his skin infection is impetigo when the doctor or nurse examines it and gives him the information; but depending upon the way he is told and the circumstances under which he is told, he may learn that accidental infections occur which do not reflect upon his

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home life, and that the doctor and nurse are his friends, that they will protect him from embarrassment in the presence of his friends and classmates, and that they will help him to get well. On the other hand, he may learn that the physician and nurse have no concern for his feelings, for they do not hesitate to tell him in the presence of other children that the infection is associated with dirt, or that he is unfit to be with his friends; he may even get the implication from conversations that he overhears in the medical room between doctor, nurse, and teacher that his home is responsible for his condition. Thus in the latter case he learns to fear the medical room and to resent these people who attack his fortress of inner security—his home.

Children and adults, who after all follow very much the same principles in learning, undergo at least three definite forms of change in each learning situation: (a) They learn the words that were spoken. (b) They learn to react emotionally to the way the total situation was handled. (c) They learn what action to take in the future to obtain any satisfactions that were present or what to do to avoid any unpleasantness that was inherent in the situation. Thus a wealth of attitudes, biases, hates, fears and prejudices are developed in every individual, and these are often found to have their origin in situations where the obvious intention of the teaching was far different.

Corollary to this principle is the fact that it is not necessary that something be done to or for an individual in order that he may learn. He frequently learns when we as teachers think we have done nothing to or for him. This is particularly true with reference to the development of attitudes. Providing a comfortable, quiet place where a mother may sit to discuss the health problems of her child with the doctor, conserving the mother's time through carefully planned appointment systems, etc., are examples of situations in which parents learn to appreciate the school health services, whereas failures to keep appointments with parents or children, advising them to go to clinics where the waiting list cannot possibly admit another person, and giving a medical examination without an explanation to the parent of what was or was not found are very likely to develop in the parents and children negative attitudes that often seem irrational to the staff.

In the light of the certainty with which learning other than that specifically intended takes place in various situations, let us examine some of the school health procedures to discover the possibilities for both positive and negative education that they present.

First of all, the initial as well as every contact of a parent or child with the physician and nurse is fraught with educational possibilities. Usually the first contact of the parent with a physician or nurse is a note either requesting the parent to take his child to a physician for an examination or inviting him to be present at the school examination of the child. Do physicians and nurses analyze such messages sent to the home to see that they accomplish the real objectives of the school health service? Is the manner in which the note is sent and the wording of it such that the parents are convinced that the request is made because the school has a vital interest in the child's health, or do the parents learn from the note that the school health service is merely carrying out a routine service without much thought as to the circumstances governing the lives of the family?

To make the point more concrete consider the following extreme example which is an actual case. "Come to

school at 9 A.M. to see me" signed "School Nurse" was hurriedly written on a scrap of paper left undated, folded, given to a boy with the command "Take that home to Mother." If such a note survives the usual vicissitudes of messages given to little boys and reaches its destination, is it likely to start a chain of events in which the parent will understand that a specific problem is to be discussed with her, will she keep the appointment with the nurse, and expect professional guidance which she may trust?

Consideration for the way in which notes are worded, who sends them home, and the method of sending them, has been experimentally shown by the New York City Study to influence the response which is obtained. One type of note resulted in a 30 per cent response from the parents. A more carefully planned note caused 85 per cent to respond to the message sent home.

The next most common experience of the parent and child with the school health service from which much positive or negative health education takes place is the visit to the medical room to see the doctor or nurse. Even the room itself can affect the parents' and children's attitudes toward, and conceptions of the school health service. If the medical quarters are clean, well lighted, well ventilated, if they have adequate room for all those who are visiting it at one time, and if there are sufficient chairs for those who must wait for short periods, other things being equal, favorable attitudes toward the health work of the school will be developed. But if, as so often happens, the physician and nurse must work in a niche in a corner, or in an empty classroom vacated because of its lack of light, the unlikely stage set will not promote a confidence in medical service or build a respect for the confidential trustworthy relationships between patient and physician which we claim we are trying to build up.

Although the physical lay-out of the medical quarters and the comforts or inconveniences that the child and parent experience while waiting in them have important influence on learning, the treatment accorded them by the doctor and nurse during the examination and accompanying interview is far more potent in determining what they learn. Are they greeted courteously? Are explanations made carefully if unexpected delays occur? Is the mother made to feel that her presence is not only welcome, but important? These are the factors that build attitudes. This, of course, is no new thought, for all school health workers have stressed the possibilities for educating both parent and child at the time of the medical examination. But in our observations of some of the examinations in various school systems, we have been led to wonder if the possibilities for learning the wrong attitudes and the wrong health behaviors from our routine procedures have been fully recognized.

Do we always recognize that the things we do not tell the parent but which are implied by our actions, lead him to form many erroneous convictions and may bring about negative attitudes toward the school health service? Perhaps an extreme situation will emphasize this point. Consider the mother who observes the examination with a stethoscope of the chest of her only child. The doctor listens—he listens again—he frowns—listens again. He has the child lie down on a couch, and listens several times, shakes his head, then listens a second time with the child standing. The mother suspecting something anxiously asks "What is the matter, Doctor?" Not wishing, without a cardiograph and more adequate examination to make a diagnosis of the heart murmur he has

heard, the doctor answers, "The nurse will tell you what to do." The nurse according to the prescribed policy gives the mother a printed slip and says in a routine fashion, "Take this slip with you and take John to your private physician. Don't worry about it." The mother looks at the slip. On it is written "cardiac" with a question mark after it.

Now more concerned than ever, having no family physician and having received no instruction in the selection of one, the mother finds a doctor's sign on the way home and decides to take Johnny there that night. The doctor after no more careful examination than was given at the school tells her that Johnny has a little heart murmur but it amounts to nothing. He further tells her that the school should have told her that it was nothing at the time.

Now what did that mother learn? First of all she learned that the school physician did not tell her all he knew. She also learned that the private physician had little respect for the school physician and the way he handled the situation. She learned to pay little attention to the advice and instruction from the school medical service. She may have learned that the school health service made her spend money for an examination that she would not have spent if she had not come in contact with it. Nor did she have the positive learning experience of finding out what a good cardiac examination is like. On the other hand, she went through an experience with considerable negative emotional content and will no doubt tell it over and over to other mothers who have children in school. Thus she becomes a negative agent of health education not because of instruction that was given her but because the school doctor in examining her child followed prescribed medical technics, but overlooked the effect of those technics on the personalities with which he was dealing.

In recounting this incident we are aware of the pressure placed upon physicians and nurses to examine a large number of children within a very short time. We are also aware of the many restrictions that policies place upon them with reference to usurping the functions of the private physicians. However, we have given the incident in the extreme to show how negative health education may result if proper consideration is not given to the attitudes and ideas that are outcomes of the procedures which we use.

If the parent has not been present at the examination and some condition needing medical attention has been found, opportunities for negative or positive education occur in the follow-up procedures. The methods used may include a note to the parent, a home visit by the nurse, a consultation in school, or contacting the child in school. Whatever the procedure, the information given should be positive and backed by sufficient reason to convince the parent that medical care is needed. All of you are familiar with the situations frequently arising during the follow-up, when the private physician or clinic disagrees with the findings of the school physician. It is recognized that disagreements in diagnosis are sure to arise, but do we consider what such disagreements teach the families with which we are working? Too often we concern ourselves only with the administrative relationships which hamper our work, and fail to recognize the effect of such situations on those who are the objects of our endeavor.

The description of these difficulties by no means suggests the solution. It does, however, point out the possibilities for negative education presented by the school health service, and what may be learned as a result of some of the

administrative procedures and policies under which the school physician and nurse must work. Although we are not in a position to suggest the proper procedures, it is our conviction that administrative policies and procedures should be influenced far more by the ideas and attitudes they engender in the parents and children than by immediate administrative exigencies of the situation.

In summary, then, there are many situations in the school health program involving the physician and the nurse that abound with teaching possibilities. If in these situations the procedures followed by the physicians and nurses bring pleasant experiences to the parents and children and satisfy their wants and needs, much good health education will result; favorable attitudes toward proper attention for the immediate health problem as well as toward the future use of medical care will be built up. If, on the other hand, the school health procedures are carried on in a routine and regulatory fashion without regard to the feelings, the wants, the comfort, and the understanding of both parents and children, much negative learning is likely to take place. Therefore, one of the contributions of the physician and the nurse to health education is to make sure that all of their procedures and methods result in positive rather than negative learning on the part of their clientele.

# Bacteriological Tests on Mechanical Dishwashers for Home Use

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MUCH attention has been given to the public health aspects of dishwashing in large establishments whereas comparatively little attention has been given the domestic problem. Since mechanical dishwashers designed for domestic purposes are now coming into use, it is necessary, for an evaluation from the public health standpoint, that scientific information be obtained concerning their effectiveness in removing bacteria from soiled dishes. Therefore, the purpose of this investigation was to determine, under controlled laboratory conditions, the amount of bacterial residue remaining on dishes after washing in two domestic dishwashers.

Machine A, the smaller, opened from the front and contained a sliding rack for holding the dishes in position during the washing procedure. Machine B opened by a hinged top and contained a double rack for utensils. Both machines had fully enameled one piece tubs with drains operated by exterior control knobs. An impeller, electrically driven, was used in both machines to throw the water across the surfaces of the dishes and remove the food soil. Water at 150° F. (65.6° C.) entered the machines through a spray mechanism situated above the dasher. Machine A held 5 quarts of water and B held 4 quarts of water during the wash period. Detergents compounded without soaps were used in the washing procedures.

## METHODS

Dishes were soiled with several types of food to simulate natural conditions. Soft boiled eggs (thoroughly minced), melted Crisco and cooked Farina were used. Suspensions of the test bacterium were added to these materials, a thorough mixing performed, and the resulting mixtures spread on the top surfaces of the dishes in a layer as nearly uniform as possible. The egg and Crisco soils were dried on the dishes for 30 minutes before washing. Dishes soiled with Farina were washed immediately after contamination. Glasses, cups, and silverware were soiled with a neutralized nucin suspension of bacteria. This material was likewise allowed to dry for 30 minutes before washing.

Calgonite (an alkaline material containing sodium hydroxide, trisodium phosphate, sodium metasilicate, and sodium hexametaphosphate) and trisodium phosphate were the detergents tested in the machines. The standard washing procedure consisted of: (1) a preliminary 10 second spray rinse with the drain open, (2) a 5 minute wash with the drain closed, (3) a 10 second spray rinse with the drain open, (4) a ½ minute rinse with the drain closed (first rinse), (5) a 10 second spray rinse with the drain open, (6) a final rinse of 1 minute with the drain closed (second rinse). At the end of this period the dishes were allowed to cool

for a few minutes before examination was begun.

Examination of the utensils was accomplished by a swabbing process. The entire front surface of all dishes such as plates, and the parts of glasses, cups and silverware normally coming into contact with the food or mouth were tested. Swabs, made up on applicator sticks, were sterilized in nutrient broth tubes (5 cc. per tube) and washed off in this broth after use. A portion of the latter was plated into suitable media to allow a count to be made. In some instances dishes in various portions of the machines were replaced with glass discs. After the soiling and washing procedures these discs were placed in large Petri dishes and washed off with melted agar. This gave an insight into the efficiency of the swabbing procedures. Veal infusion agar was used to test for *Eberthella typhosus* and *Staphylococcus aureus* while blood agar was used for streptococci. In testing for *Mycobacterium tuberculosis*, inoculations were made subcutaneously into the groin of guinea pigs. All plates were incubated at 37°C. for 48 hours.

## EXPERIMENTAL

Food soil, to which a suspension of *Bacterium typhosum*, *Staphylococcus aureus* or *Streptococcus pyogenes* had

TABLE I

*Summary of Results Obtained by Machine Washing with 0.3 Per cent Calgonite*

| *Bacterium* / *Food Soil* / *Machine* | *Staphylococcus aureus* Egg | | *Staphylococcus aureus* Crisco | | *Eberthella typhosus* Egg | | *Streptococcus pyogenes* Egg | | *Staphylococcus aureus* Farina |
|---|---|---|---|---|---|---|---|---|---|
| | *A* | *B* | *A* | *B* | *A* | *B* | *A* | *B* | *A* |
| *Sterile dishes* * | | | | | | | | | |
| Per cent sterile after: | | | | | | | | | |
| wash | 10 | 17 | — | — | — | — | — | — | — |
| 1st rinse | 15 | 25 | — | — | — | — | — | — | — |
| 2nd rinse | 32 | 70 | 63 | 56 | 72 | 99 | 51 | 89 | 99 |
| Average number of bacteria per dish remaining contaminated after: | | | | | | | | | |
| wash | 14 | 16 | — | — | — | — | — | — | — |
| 1st rinse | 4 | 5 | — | — | — | — | — | — | — |
| 2nd rinse | 6 | 3 | 7 | 2 | 2 | 0 | 4 | 3 | 0 |
| Maximum number of bacteria per dish remaining contaminated after: | | | | | | | | | |
| wash | 17 | 20 | — | — | — | — | — | — | — |
| 1st rinse | 8 | 8 | — | — | — | — | — | — | — |
| 2nd rinse | 7 | 8 | 32 | 8 | 5 | 1 | 8 | 3 | 1 |
| *Soiled dishes* | | | | | | | | | |
| Per cent sterile after: | | | | | | | | | |
| wash | 10 | 20 | — | — | — | — | — | — | — |
| 1st rinse | 20 | 70 | — | — | — | — | — | — | — |
| 2nd rinse | 30 | 77 | 50 | 61 | 60 | 90 | 50 | 90 | 79 |
| Average number of bacteria per dish remaining contaminated after: | | | | | | | | | |
| wash | 412 | 502 | — | — | — | — | — | — | — |
| 1st rinse | 8 | 16 | — | — | — | — | — | — | — |
| 2nd rinse | 7 | 8 | 11 | 10 | 6 | 4 | 4 | 4 | 5 |
| Maximum number of bacteria per dish remaining contaminated after: | | | | | | | | | |
| wash | 2,400 | 3,200 | — | — | — | — | — | — | — |
| 1st rinse | 16 | 32 | — | — | — | — | — | — | — |
| 2nd rinse | 12 | 28 | 21 | 56 | 10 | 4 | 20 | 6 | 16 |

* Sterile dishes were introduced into machine with soiled dishes.
— indicates no tests made.

been added, was put on sterile dishes and the latter washed in the machines. Glasses, cups, and silverware were not soiled at this time, but they, along with other dishes, were tested by the swab method at the end of the wash period. This was done to determine the amount of transfer that occurred from the contaminated to the sterile dishes. Tests were also made to determine the efficiency of the wash and rinse procedures. In such tests the dishes were swabbed at the conclusion of the 5 minute wash period, after which the experiment was begun anew, and tests made at the end of the first and second rinses in a similar fashion. As would be expected the first rinse is quite effective in removing contaminating organisms. Calgonite, in 0.3 per cent concentration by weight, was used in obtaining the results tabulated in Table I. The heading, " Sterile Dishes," in that table is used to indicate glasses and cups that were put into the washer in a sterile condition along with the soiled dishes.

In those experiments in which glass discs were substituted for dishes, the results showed the swab method to be approximately 50 per cent efficient in recovering organisms from contaminated surfaces when only small numbers were involved. Thus, those dishes listed as " Sterile " in Table I and subsequent tables may or may not be in this condition. However, the majority of the results in the literature are based upon a swab method of recovery. These data are in agreement with those of Fellers, Levine, and Harvey.[1]

*Staphylococcus aureus* withstood the washing procedure better than either *Streptococcus pyogenes* or *Eberthella typhosa*. According to Devereux and Mallman,[2] Calgonite is quite effective as a germicide against *Escherichia coli* but it has little or no effect on *Staphylococcus aureus*. Our results support this observation. When bactericidal tests were made at 140° F. (60° C.) by *in vitro* methods, the effect of heat overshadowed any possible effect of the detergent and all organisms of the three types tested were killed in from 5 to 7 minutes. Varying the degree of hardness of the water with either detergent gave no alteration of these results. However, in both the A and B machines the water was cooled to such an extent by the dishes that only the second rinse water in machine A reached 141.8° F. (61° C.), while that in the larger machine B only reached 135° F. (57.2° C.). Tests made at a temperature of 122° F. (50° C.) showed viable organisms present at the end of 10 minutes.

When trisodium phosphate was substituted for Calgonite and a large number of washings made over a period of time, with no sterilization of the machine or dishes during such a period, a noticeable film formed on the

TABLE II

*Viable Bacteria on Cups, Glasses, and Silverware Soiled with a Neutralized Mucin Suspension of Staphylococcus aureus and Washed by the Standard Procedure in Machines A and B*

| *Type of Utensil* | *Per cent Dishes Sterile* | | *Average Number of Bacteria per Dish Remaining Contaminated* | | *Maximum Number of Bacteria per Dish Remaining Contaminated* | |
|---|---|---|---|---|---|---|
| | *A* | *B* | *A* | *B* | *A* | *B* |
| Glass | 77 | 74 | 7 | 6 | 24 | 20 |
| Cup | 80 | 72 | 5 | 7 | 8 | 16 |
| Knife | 83 | 61 | 6 | 6 | 8 | 20 |
| Fork | 92 | 80 | 8 | 5 | 8 | 8 |
| Spoon | 83 | 80 | 6 | 4 | 8 | 8 |

utensils. This was especially noticeable on the glasses. Bacteriological methods in which the film was completely broken up did not show that there was any noticeable accumulation of bacteria as has been reported by Hall,[3] Hall and Schwartz,[4] and Cox.[5] These workers were not concerned with a specific bacterium of a pathogenic type, so it is entirely possible that other types of bacteria normal to air, water, etc., would accumulate in such a film. It is doubtful, though whether they would be capable of growing in a film of this type. Recently, Hall[6] reported on the use of a milk culture of *Staphylococcus aureus* as a soiling agent. The dishes were washed with a trisodium phosphate detergent and the film was definitely found to contain bacteria. However, to quote from Halvorson, Bayliss, Ordall, and Wilson[7]: "The type and extent of the deposits formed depends upon the water hardness and temperature, the type of soil, the nature of the surface being cleaned, the soap and buffers used, the washing procedure, and other such factors."

In order to gain an idea as to the relative efficiency of the machines in contrast to hand washing procedures, dishes were soiled with bacteria-food mixtures and washed by hand, using a common brand of flake soap. The wash and rinse waters were adjusted to an initial temperature of 120° F. (48.9° C.). The swab method of testing was used. All the dishes remained contaminated, but to a varying extent. As an example, there was an average of 26 bacteria for each of the soiled dishes after rinsing and air drying in contrast to 11 when identical mixtures were used for soiling and the dishes washed in a machine. Sterile cups, run through the wash and rinse water, retained an average of 18 organisms in contrast to 7 when washed in a machine.

Cups, glasses, and silverware were soiled with *Staphylococcus aureus* suspensions in neutralized mucin and washed by the standard procedure using Calgonite (see Table II). In experiments of this type all other dishes were placed in the machines in a sterile condition so that the transfer from plates, etc., as previously shown in Table I, would be eliminated. The maximum number of bacteria per utensil was never greater than 24 when tested by the swab procedure. A large majority of the dishes appeared to be sterile.

In view of the possible dissemination of tubercle bacilli by contaminated utensils and considering the resistance of this organism, it was thought that results of some significance could be obtained by using such an organism in the food soil. The H37 strain of the human tubercle bacillus was grown on Lowenstein's medium, ground in a mortar with sterile reagent sand, and mixed with finely divided egg material. Dishes were soiled in the usual fashion. Tests were made of the preliminary spray rinse, of the main wash water, and of the dishes. In the latter tests 5 of one type of dish were swabbed with a single swab. The swab was rinsed in 4.0 cc. of sterile saline after each dish was examined. Guinea pigs were inoculated subcutaneously into the groin with the test material. The materials containing Calgonite were neutralized before being injected. Results are given in Table III.

From Table III it is apparent that the organism causing human tuberculosis can withstand the washing procedure and still be present in small numbers on eating utensils.

Although both machines had fully enameled one piece tubs, there were certain regions where accumulation of food soil and bacteria might occur. These regions were checked by a process of cumulative washing in

TABLE III

*Recovery of Mycobacterium Tuberculosis from Dishes Washed in Machines A and B with Calgonite Detergent*

| *Material* | *Inoculum* | *Number of Guinea Pigs Used for Each Machine* | *Number of Guinea Pigs with Tuberculosis* | |
|---|---|---|---|---|
| | *cc.* | | *A* | *B* |
| Preliminary spray rinse | 0.2 | 10 | 8 x | 10 |
| Wash water | 1.0 | 10 | 10 | 9 x |
| Swab material from: | | | | |
| Plates | 1.0 | 2 | 2 | 1 |
| Saucers | 1.0 | 2 | 1 | 0 |
| Bread dishes | 1.0 | 2 | 0 | 1 |
| Salad dishes | 1.0 | 2 | 1 | 1 |
| Sauce dishes | 1.0 | 2 | 2 | 2 |

x = others died within 7–10 days after inoculation from other causes.

which dishes were washed several times daily for periods of 1 week with no sterilization or cleaning of the machine during the period. Egg soil containing *Staphylococcus aureus* was used in these tests. Machine A showed some accumulation beneath a door stop of rubber and around a perforated plate over the drain valve. In machine B the greatest and only accumulation of importance was on a portion of the groove in which the lid gasket rested. However, there was no evidence of bacterial growth or odors in the sections where accumulation occurred.

## SUMMARY

Two mechanical dishwashers designed for domestic purposes were subjected to rigidly controlled tests for the removal of bacteria from dishes contaminated with food soils consisting of one of the following: egg, fat (Crisco), wheat cereal (Farina), and neutralized mucin. The food soils were inoculated with one of the following bacteria: *Staphylococcus aureus, Eberthella typhosa, Streptococcus pyogenes,* and *Mycobacterium tuberculosis.* For comparative purposes dishes similarly soiled were washed by hand in water to which a common brand of soap had been added.

Dishes washed in the two machines were not sterile but had fewer residual bacteria than those washed by a hand procedure. The numbers of residual bacteria are apparently reduced by a mechanical washing-away action and not by the temperautre of the water or any great germicidal effect of the detergents. According to Behrend,[8] utensils yielding a count of from 1 to 10 bacteria may be classified as excellent, from 11 to 30 good, from 31 to 50 fair, from 51 to 100 poor, and more than 100 bad. On this basis the machines on the average, when used according to standard procedures, gave excellent results.

The ideal dishwasher from the public health standpoint is one which will remove all pathogenic microörganisms from dishes. At present certain technical difficulties interfere with the achievement of that goal.

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# Active Immunization Against Diphtheria

## The Efficacy of Several Methods Used in a City of Medium Size

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DURING the last five decades there has been a decline in the mortality from diphtheria. Figure I, beginning with the year 1870, shows Cincinnati's crude mortality in children under 10 years of age. A peak was reached in 1890.

Morbidity records are less accurate than mortality statistics, but the general trend of diphtheria morbidity in Cincinnati during the last three decades has been downward. There was an acceleration of this downward trend in 1928 and 1929 when the first local large scale immunization program was inaugurated. This result was encouraging although the decline was not greater than that which had occurred at previous times when active immunization had not been employed. Toxin antitoxin was the first antigen used. Sometimes 3 and sometimes 4 injections were given at intervals of 1 week. Two injections of formolized toxoid, 1 week apart were substituted in 1932. In 1934, the single injection of alum precipitated toxoid was given, and in 1936, this was changed to 2 injections of alum precipitated toxoid at an interval of 4 weeks. Innovations in antigen and the number of injections have been introduced so rapidly that the merits of the various methods have not been completely evaluated.

Efforts should be made to determine accurately whether the decline in diphtheria has been produced by control measures or by unknown factors governing the natural cycles of the disease. Lucia and Welke [1] made a comparison between mortality trends in Providence, R. I., where there had been a vigorous campaign for immunization against diphtheria, and San Francisco, Calif., where there had not been such an active campaign. The trends in both cities were similar. Woods [2] showed that in England the decline in mortality from diphtheria was similar to that from scarlet fever although no large scale immunization measures against scarlet fever had been carried out. On the other hand, experience in institutions, Young and Cummings,[3] in schools, Park,[4] and in health districts, Gundel,[5] indicates that active immunization has helped in reducing the incidence of diphtheria.

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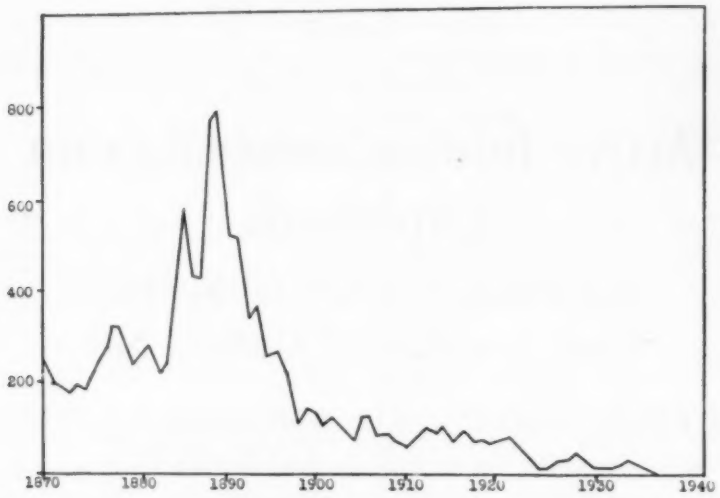



Diphtheria Mortality for Cincinnati, Ohio, Rates per 100,000 Under 10 Yrs. of Age - 1870 - 1937.

Figure I

EPIDEMIOLOGY OF DIPHTHERIA

Resident cases for the years 1932–1937, were studied. The population figures used were those obtained in a local census in the year 1935.

*Distribution by Season, Sex and Age*—In Cincinnati, the epidemiology of diphtheria has generally conformed to the usual pattern. The peak incidence has been reached in late autumn and early winter. In the earlier years of life, both sexes have been equally affected. In the few cases which have occurred after 20 years of age, there has been the well known preponderance of the disease in females because of their close association with children. In relation to age, the greatest incidence of cases and deaths has been in the late preschool and early school years.

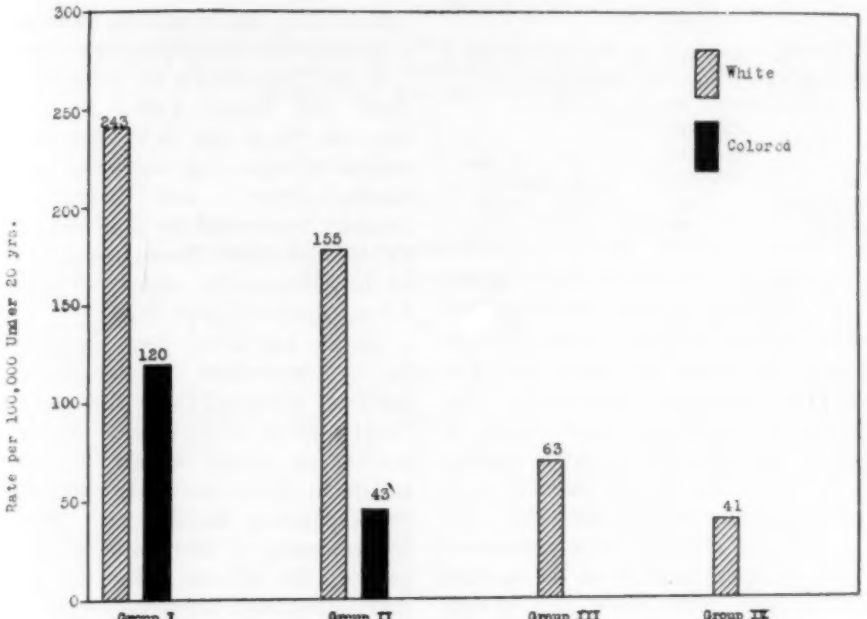
*Distribution by Economic and Racial Groups*—These two topics may be appropriately considered together. Cincinnati's area has been divided into 107 census tracts, and these in turn fall into 4 economic groups, based on average rentals. The populations of the 4 groups are approximately equal.

TABLE I

*Distribution of Diphtheria in White and Colored in Relation to Economic Level*

| | *White* | | | *Colored* | | |
|---|---|---|---|---|---|---|
| *Economic Group* | *Population Under 20 Yrs. of Age in 1935* | *Total Resident Cases Under 20 Yrs. of Age in 1932–1937* | *Average Yearly Rate per 100,000 Under 20 Yrs. of Age* | *Population Under 20 Yrs. of Age in 1935* | *Total Resident Cases Under 20 Yrs. of Age in 1932–1937* | *Average Yearly Rate per 100,000 Under 20 Yrs. of Age* |
| I (Lowest) | 27,631 | 403 | 243 | 11,075 | 80 | 120 |
| II | 30,906 | 288 | 155 | 5,024 | 13 | 43 |
| III | 30,925 | 117 | 63 | 1,938 | 8 | 69 * |
| IV (Highest | 30,481 | 75 | 41 | 460 | 2 | 72 * |

* Omitted in preparation of Figure II because of questionable validity.




Distribution of Diphtheria by Economic Groups - White and Colored.
Morbidity Rates per 100,000 under 20 years of age - Six Year Average.

(Colored Rates have not been shown for Groups III and IV because of questionable validity.)

Figure II

Group I is the lowest and Group IV is the highest in respect to economic well-being.

Table I and Figure II show that diphtheria like tuberculosis, is a disease associated with poverty. It is also indicated that the white race is more susceptible to diphtheria than the colored. A comparison of the total white and colored populations under 20 years of age would not have been valid because most of the colored population is in the lower economic groups.

The preponderance of diphtheria in economic Groups I and II cannot be attributed to neglect of inoculations. The Health Department and other agencies have provided free inoculations in these groups while Groups III and IV have been cared for by private physicians.

### DIPHTHERIA DESPITE INOCULATION

Cases of diphtheria occur even in those inoculated. The term inoculation rather than immunization is used for the reason that the latter implies that the desired result has been obtained. From the data available, it is impossible to tell which of the standard antigens has been most efficacious. Cases of diphtheria have occurred after the administration of all of them. Many occurred within a year after the administration of the antigen. The failure to prevent diphtheria in these instances, therefore, cannot be attributed to loss of immunity over a long period of time.

The fact that attempts to immunize against diphtheria sometimes fail is not of recent discovery. Park [4] pointed out that even in children with negative Schick tests, naturally or artificially produced, the incidence of diphtheria was decreased but the disease was not entirely eliminated.

Do those who have contracted diph-

TABLE II

*Cases Under 20 Years of Age in Which a Definitely Positive or Negative History of Previous Inoculation Could Be Obtained*

| | Cases | Deaths | Fatality Rate Per cent |
|---|---|---|---|
| Not Inoculated | 474 | 22 | 4.64 |
| Inoculated | 238 | 11 | 4.62 |

theria despite inoculation have milder cases? Fatality rates in non-inoculated and inoculated children (under 20 years of age) are shown in Table II. The difference was not significant. The only cases considered were those in which it was possible to get a definite yes or no answer to the question concerning previous inoculation. All cases in inoculated children were considered in one group regardless of the antigen used because there were not enough cases to consider mortality in respect to each type of antigen. Cases developing within 6 weeks after completion of inoculation and cases in which the full course of inoculations had not been completed were considered as non-inoculated.

When diphtheria has developed in previously inoculated children, physicians have not delayed in giving antitoxin. The average interval between onset of sickness and administration of antitoxin was slightly shorter in inoculated than in non-inoculated children.

The incidence of diphtheria in inoculated and non-inoculated children of the same race and comparable economic levels could not be calculated because of lack of accurate data concerning the number and distribution of the inoculations. Of the cases of diphtheria in the 238 previously inoculated children 195 (81.9 per cent) were in the low economic groups, I and II. The distribution of the cases in inoculated children was similar to the general distribution where 784 (79.5 per cent) of the 986 cases in children were in the same low economic groups.

## ARTIFICIAL AND NATURAL IMMUNITY AS MEASURED BY THE SCHICK TEST

It has been shown by Guthrie, Marshall, and Moss,[6] Park,[4] and others that the Shick test is a fairly reliable method of measuring immunity to diphtheria. Jensen[7] and Brandon and Frazer[8] have recently and more accurately determined the approximate level of blood antitoxin which is necessary for a negative Schick test.

As an individual goes through life, he has exposures to the diphtheria bacillus, frequently in the city, less frequently in rural areas. Active immunization should be carried out early in life in order that early exposures to the diphtheria bacillus may stimulate higher levels of immunity rather than produce the disease. The duration of artificial active immunity will depend to some extent upon the frequency of subclinical infections. Therefore, in comparing the duration of immunity conferred by various types of inoculations, strictly comparable groups must be studied. Rural and even suburban populations must not be compared with urban populations because the frequent subclinical infections in crowded areas produce a prolongation of artificial immunity that cannot be expected in more sparsely populated areas.

In this Shick test survey two neighboring suburbs, M. and E.E., were studied during the school year 1935–1936. Both suburbs were relatively homogenous areas, intermediate between rural and urban. A control group and groups of children who had received various numbers of injections of various antigens without preliminary Schick tests were used. The findings in the various groups were not exactly comparable because the intervals of time between the administration of the various antigens and testing were not always the same. Nevertheless, it was possible to reach certain conclusions. The results are given in Table III.

TABLE III

*Schick Tests in a Relatively Homogenous Suburban Area—Groups of Children Who Had Received Various Numbers of Injections of Various Antigens and a Group Who Had Received None*

| | Children in Group | Schick Positive | | Schick Negative | | Interval between Inoculation and Schick Test in Years | Mean Age in Years at Time of Schick Test |
|---|---|---|---|---|---|---|---|
| | | Number | Per cent | Number | Per cent | | |
| Non-Inoculated (Controls) | 544 | 359 | 66 | 185 | 34 | | 11.22 ± .09 |
| Toxin-Antitoxin (4 Injections) | 208 | 60 | 29 | 148 | 71 | 6 | 10.80 ± .09 |
| Toxin-Antitoxin (3 Injections) | 735 | 253 | 34 | 482 | 66 | 4–7 | 10.93 ± .08 |
| Ramon Toxoid (2 Injections) | 650 | 297 | 46 | 353 | 54 | 2–3 | 7.66 ± .06 |
| A. P. Toxoid (1 Injection) | 219 | 45 | 21 | 174 | 79 | 1 or less | 6.50 ± .15 |
| Total Tested | 2,356 | | | | | | |

Schick test readings which gave prolonged nonspecific reactions (about 5 per cent) were not included.

Each of the groups of inoculated children showed a definitely higher level of immunity than the control group. This was particularly significant because the controls were older and therefore had a higher level of natural immunity. The relatively good results in the group which received one injection of alum precipitated toxoid may have been due to the short interval of time between inoculation and testing. The somewhat poorer results in the group which received two injections of formolized toxoid may have been due to the younger age of the members of that group, or to the fact that they received only 2 injections 1 week apart. The results in the group which received toxin-antitoxin were relatively final and represented the resultant immunity status after primary stimulus and natural immunization had reacted over a substantial period of years. There was no significant difference between the immunity levels of those who received 3 injections of toxin-antitoxin and those who received 4.

## DISCUSSION

No actively immunizing agent provides perfect protection against diphtheria. This is not surprising in view of the fact that the disease itself does not provide complete protection against subsequent attacks.

The results of this survey indicate that any one of the standard antigens properly given produces an immunity level higher than the level in similar controls. There is no clear-cut evidence that the immunity level produced by any particular antigen is higher or more lasting than that produced by the others. Much accurate data must be accumulated over a long period of years before the final answer concerning the best method can be given.

The findings of Wells, Graham, and Havens,[9] Friedman,[10] Graham, Murphree, and Gill,[11] and Saunders[12] confirm the opinion that inoculation with alum precipitated toxoid is the best single injection method of immunization. We have, therefore, continued its use, supplemented by a second injection. It is desirable to do as complete a job as possible with the first injection because indifferent and care-

less mothers sometimes fail to bring their babies back for subsequent treatments.

There are several reasons for giving a second injection of antigen. First, the reports of Bousfield [13] cast doubt upon the efficacy of the single injection, even of alum precipitated toxoid. Second, the work of Pansing [14, 15] indicated that the duration of immunity is longer in children who have received two injections of alum precipitated toxoid. This is particularly significant because Pansing has worked in a rural area where efforts to produce immunity have not been helped along by frequent subclinical infections. Third, the animal experiments of Glenny and Südmersen [16] indicate the value of secondary stimulus. If their observations are to be extended to human beings, the interval between injections should be moderately long—at least 4 weeks.

Since May, 1936, we have been using two injections, 1 cc. each of alum precipitated toxoid at an interval of 4 weeks. This is essentially the procedure which Park [17] has advocated. A few cases of diphtheria have developed even after this treatment, but it is too soon for an evaluation of the results. Schick tests on a little over 100 preschool children in suburb M, indicate that 90 per cent are Schick negative 3 months after the second injection of alum precipitated toxoid.

## CONCLUSIONS

1. The morbidity and mortality from diphtheria are declining. Artificial immunization has probably accelerated these declines.

2. Several injections of antigen give better immunization than a single injection. In the case of toxin-antitoxin used in a suburban area there is a leveling off after 3 injections. Four injections do not give significantly better results.

3. Cases and deaths from diphtheria occur even after inoculation. So far, there is no definite evidence that such cases are less severe than cases in non-inoculated children.

4. Diphtheria affects predominantly the children of poor white parents. In the control of the disease, the improvement of living and housing conditions is important.

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# Similarity of Chronic Infections With Salmonella Typhimurium and Tuberculosis in Guinea Pigs

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THE occurrence of Salmonella infections among guinea pigs has been recognized for many years. Smith and Stewart [1] first described the condition in the United States and gave it the name "pseudo-tuberculosis." The resemblance between this infection and that produced by tubercle bacilli is quite close as has been repeatedly emphasized. The condition has usually attracted attention, however, because of the increased mortality it produces in guinea pig herds rather than because of the confusion which may arise when such diseased animals are used for diagnostic purposes. In view of the increasing use of guinea pigs for the detection of subclinical and minimal tuberculosis in man, it appears desirable to point out the difficulties which may arise from this source.

"Pseudo-tuberculosis" occurs both as epizoötics with, at times, an appalling mortality (Wherry,[2] Plague Commission,[3] Petrie and O'Brien,[4] Howell and Schultz,[5] Gheorghi and Barth [6]), and as enzoötics (Thomas,[7] Edington,[8] Duthie and Mitchell,[9] Walker and Sweeney [10]). The transition of an epizoötic into an enzoötic has been described by Nelson and Smith [11–14] in a series of papers. The outbreak to be discussed by us was apparently an enzoötic which was activated into an epizoötic of mild degree.

In this laboratory approximately 50 guinea pigs are inoculated each week with material obtained from patients thought to have tuberculosis. These animals are 6–8 weeks old at the time of inoculation, and are supplied to us as they are required. They are placed in a single large pen when received. After injection, they are placed in individual cages in another room where they remain for 6 weeks, when they are killed and autopsied to determine whether they have developed any lesions of tuberculosis. Ordinarily, we expect about 6–8 per cent of the inoculated animals to die of intercurrent infection during the 6 weeks' observation. In the early part of August, 1938, following a severe heat wave, the mortality rose to 19 per cent. At first these deaths were attributed to the heat as no lesions could be found at autopsy. Shortly afterward we began to find an occasional animal which presented all the gross pathological changes seen in guinea pig tuberculosis of the artificial variety, but in which no acid-fast bacilli could be found in the routine smears made from the caseous lesions. Microscopic sections of the spleens showed a histological picture identical with that of tuberculosis. On more thorough bacteriological investigation we were able to demonstrate Gram-negative bacilli.

TECHNIC

Each guinea pig was opened with clean but not sterile instruments. Smears were made from any accumulation of fluid in either the abdominal or thoracic cavities. Cultures of the blood, urine, and bile were made with aseptic precautions into 1 per cent glucose broth. Several fecal pellets, were removed from the lower bowel, macerated in a 30 per cent glycerin–0.5 per cent lithium chloride mixture, and a heavy inoculation smeared over the surface of a Blair-Wilson bismuth sulfite medium plate. The spleen was removed, passed through a flame to sterilize the outer surface, and transferred to a sterile mortar, ground to a pulp, a thick emulsion made with sterile normal saline, and finally inoculated into 1 per cent glucose broth. All cultures were incubated for 48 hours and those showing growth were subcultured on a litmus-lactose agar plate to test the purity of the culture. Colonies from this plate were then inoculated into the various test sugars and agglutinated by a specific serum (strain 1 serum).

BACTERIOLOGY

One hundred and forty-eight animals were studied, 48 of which were found to be infected. Seventy-eight cultures were completely identified; 39 from the spleen, 25 from the feces, 9 from the blood, 3 from the urine, and 2 from the bile. All of these were identical culturally and serologically with the original culture isolated from a caseous abscess in a spleen and referred to in this paper as strain 1. This strain was used to immunize a rabbit to produce an immune serum with a titer of 1–10,000 for the homologous organism.

Cultures of the heart blood were always found to be positive in animals dying from the disease—either acute or chronic. In 4 animals killed, and found at autopsy to be suffering from the disease, blood cultures were negative. Urine and bile cultures were not made routinely, but the urine was found to be infected in 4 of 8 diseased animals, the bile in 2 out of 6. In 32 infected animals, cultures of both the spleen and feces were made; in 15 both were positive; in 13 the spleen was positive while the feces were negative; in 4 the feces were positive but the spleen negative. Whether these latter animals were passive carriers cannot be proved, but it is certainly suggestive.

Previous workers have shown that the infecting organism is either *Salmonella typhimurium* (Edington,[8] Petrie and O'Brien,[4] Nelson [13]), or *Salmonella enteriditis* (Duthie and Mitchell,[9] Plague Commission,[3] Thomas [7]). Much of the earlier work has to be discarded because of the recent advances in classifying members of this group. Wherry [2] reported an organism which he called *Bacillus pestis caviae,* but which Jordan [15] has classified as *Salmonella enteritidis.* Jordan [15] and Raebiger and Lerche [16] state that *Salmonella typhimurium* is much more common than *Salmonella enteritidis.* Tenbroeck [17] found the reverse to be true.

The organism isolated from our cases was a pleomorphic, Gram-negative bacillus varying from short coccoid forms to large bacillary ones. The smaller forms stained bipolar or barred and were the type usually seen in smears from exudates, especially if the organisms were numerous. The larger solidly staining bacillary forms were occasionally seen in animals and always in culture. No capsule was demonstrated. The organism was actively motile. Growth was profuse on all media. On plain agar the colonies were round, somewhat opaque, white, with a smooth edge. Good growth occurred on the Blair-Wilson medium, the colonies being lusterless and dull gray with considerable blackening of the medium. Lactose and sucrose were not fermented. Acid and gas were formed in glucose, mal-

TABLE I

*Agglutination Titers of Various Members of the Salmonella Group With Their Respective Sera*

| | *S. Paratyphi-B Serum* | *Strain I Serum* | *S. typhimurium Serum* | *S. cholerae-suis Serum* |
|---|---|---|---|---|
| Strain I | 1–6,400 | 1–10,000 | 1–2,500 | 1– 400 |
| *S. typhimurium* | 1–3,200 | 1– 6,400 | 1–2,500 | — |
| *S. paratyphi*-B | 1–6,400 | 1– 6,400 | 1–1,600 | — |
| *S. cholerae*-suis | — | 1– 400 | — | 1–12,800 |
| *S. enteritidis* | — | 1– 100 | — | — |

tose, mannite, sorbitol, rhamnose, dulcitol, xylose, and mannose in 18 hours. After 4–7 days' incubation the dulcitol and sorbitol media became secondarily alkaline using brom thymol blue as an indicator. The methyl red test was positive in 24 hours. Indol was not formed after 4 days' incubation. Litmus milk was acid after 18 hours, becoming strongly alkaline after 3–4 days' incubation.

This bacillus was identified as *Salmonella typhimurium* from the serological tests. The tests were made with known members of the Salmonella group in stock and with antisera previously prepared against these stock strains. As most of the antisera were old, the titer was apt to be rather low. This was particularly true of the *Salmonella typhimurium* serum (titer 1–2,500). Tables I and II represent a typical protocol of the serological experiments. All agglutination tests were incubated at 37° C. for 2 hours and left overnight at 5° C. Absorption tests were placed in a 50°C. water bath for 2 hours and then placed in the icebox overnight. Usually one massive absorption was sufficient to remove all the agglutinins for the absorbing organism. If this did not suffice, a second absorption was done using a smaller quantity of organisms. Two absorptions were the most ever required.

The pathogenicity of this organism for guinea pigs was readily proved. Three strains were used, one isolated from the spleen of an animal with chronic disease, one from the blood stream of an animal dying of the acute disease, and the third from a culture of feces. Subcutaneous and intraperitoneal injection of 10–20 million organisms into 6 animals produced death in 18 hours in 3, 1 died on the 4th day after injection, and 2 on the 5th day. The changes found at autopsy were the same regardless of the site of inoculation. There was considerable injection of the blood vessels over the abdominal musculature and beneath the skin. The peritoneum and pleura were markedly injected. Both the peritoneal and pleural cavities contained 3–10 cc. of thin, slightly yel-

TABLE II

*The Results of Reciprocal Agglutinin Absorption Tests*

| *Organism Tested* | *S. Paratyphi-B Serum Absorbed With Strain I Organisms* | *S. typhimurium Serum Absorbed With Strain I Organisms* | *Strain I Serum Absorbed With S. paratyphi-B Organisms* | *Strain I Serum Absorbed With S. typhimurium Organisms* |
|---|---|---|---|---|
| Strain I | 0 | 0 | 1–400 | 0 |
| *S. typhimurium* | 0 | 0 | 1–200 | 0 |
| *S. paratyphi*-B | 1–200 | 0 | 0 | 0 |

0 = No agglutination of the organisms with the absorbed sera diluted 1–100

lowish, albuminous fluid in which bacilli were easily demonstrated. In the animals which died within 24 hours, the viscera showed no changes, gross or microscopic, beyond an intense, hemorrhagic congestion and many fresh fibrinous adhesions about the spleen, over the dome of the liver, and occasionally between the loops of small intestine. In the 3 guinea pigs which lived for 4–5 days, the liver and spleen were found to have enlarged to about twice the normal size. In one animal dying on the 5th day many tiny white nodules were found throughout the spleen. Microscopically these were tiny areas of endothelial proliferation identical with those found in the naturally infected guinea pigs. The organisms were readily recovered from the blood, spleen, urine, and feces of these experimental animals.

PATHOLOGY

Two types of gross anatomical lesions, with all degrees of gradation between, were seen in the guinea pigs which were naturally infected. Some animals died of an acute hemorrhagic disease with lesions exactly like those found in our inoculated guinea pigs. In about half the spleen showed a slight but definite enlargement. Such animals in the cages would eat well and gain in weight until about 24 hours before death when they would appear listless with ruffled fur. In a few the time between the onset and death was so short that no evidence of illness was seen.

Most of our animals died of the chronic form of the disease. They appeared listless and had ruffled fur for 4–7 days before death, but continued to eat well until they were moribund, and the loss of weight was minimal. Indeed, in those guinea pigs killed routinely at the end of 6 weeks, such animals often appeared fatter and larger than their normal brethren.

At autopsy these animals had no injection of the superficial bood vessels. The peritoneal and pleural cavities contained no fluid. Old fibrinous adhesions about the spleen and between the upper loops of small bowel and the stomach were common. The mesenteric lymph glands were, as a rule, only a little enlarged. The spleen was always enlarged, at times attaining a size of 4 x 2 cm. as against an average normal size of 2½ x 1¼ cm. The spleen always contained yellowish-white, firm nodules, ranging in size from those barely visible up to 2–3 cm. across and varying in number from 1 to 100 or more. At times the spleen contained definite abscesses, 5–10 mm. across, in the pus of which the Salmonella organism was easily demonstrated. Nodules similar to those occurring in the spleen were occasionally found in the liver and over the surface of the peritoneum. No gross pathological changes were found in the other organs.

Histologically the most typical lesions are seen in the spleen and consist of endothelial proliferation. This begins in the center of the lymph follicles and gradually increases until the entire follicle has been replaced by large endothelial cells, which form a solid, compact mass which may replace all or most of the normal splenic tissue. In the center of the larger nodules necrosis of the endothelial cells occurs together with the infiltration of many polymorphonuclear leucocytes. In the largest nodules the center is entirely composed of pus cells and cellular débris. The endothelial cells remaining outside of this central necrotic area become elongated and take on the appearance of fibroblasts. These cells form a sort of capsule so that grossly the entire nodule may be separated from the surrounding splenic tissue. The " capsule " always contains a few lymphocytes. Aside from some slight engorgement of the capillaries, there are no other lesions in the spleen.

The nodules in the liver and on the peritoneal surface apparently arise through the same process as those in the spleen. Accumulations of large mononuclear cells, having the appearance of endothelial cells, occur in these areas. These do not seem to arise from lymphoid follicles. In the largest nodules central necrosis and polymorphonuclear leucocytic infiltration occurs. The capillaries of both the liver and kidney are distended. No areas of endothelial cell proliferation were found in any organ other than those mentioned. Lesions in all of the organs have been reported at one time or another by other authors (Edington,[8] Raebiger and Lerche,[10] Howell and Schultz,[5] Thomas,[7] Duthie and Mitchell,[9] and Nelson and Smith [11]).

Differentiation between the chronic form of "pseudo-tuberculosis" and true tuberculosis, although usually not difficult, at times may be impossible. Histologically there is no difference between them. The tuberculous nodules produced by inoculation of guinea pigs arise from the proliferation of the endothelial cells with a secondary central necrosis and infiltration by polymorphonuclear leucocytes. Giant cells and typical tubercle formation as seen in human tuberculosis do not occur in the guinea pig. Care should be taken not to confuse the megalokaryocytes occasionally found in normal guinea pig spleens with tuberculous giant cells.

Differentiation is best made from the gross lesions. The characteristic feature of both diseases is the firm, yellowish-white nodules usually found in an enlarged spleen and occasionally distributed through the liver and over the peritoneum. Large caseous abscesses, which are not uncommon in "pseudo-tuberculosis," have never been found in our animals with true tuberculosis. Moreover, fibrinous abdominal adhesions appear to be more common in animals infected with *Salmonella typhimurium* than in those with tubercle bacilli. In tuberculosis the mesenteric lymph glands are large, caseous, and there is considerable matting together and thickening of the omentum. In "pseudo-tuberculosis" the glands are discreet and only slightly enlarged. However, considerable glandular enlargements may, in rare instances, be observed in "pseudo-tuberculosis" while the reverse picture of small discreet lymph glands and splenic nodules is not infrequently observed in our tuberculous pigs. Differentiation of the two conditions must depend on demonstrating the causative organism in the lesions. Routine smears from suitable caseous areas should be made in every instance, and no diagnosis of tuberculosis should be made unless acid-fast bacilli are found in such smears.

### EPIDEMIOLOGY

Although shedding of the organisms through the intestinal tract was early recognized, attempts to infect guinea pigs by feeding have been unsuccessful or have succeeded only by the use of enormous numbers of organisms. Such experimental animals may show the organism in their feces but fail to develop any clinical disease (Smith and Nelson,[12] Petrie and O'Brien,[4] Friedlander and Hertert [13]). Nelson [14] has shown that healthy animals, placed in contact with sick ones under natural conditions, will become infected. However, Petrie and O'Brien [4] and Duthie and Mitchell [9] were unable to infect animals by this method.

As none of our guinea pigs ever showed lesions in the lungs, it would seem as though the infection were transmitted by way of the alimentary canal. Bacteria were found to be discharged from the sick animals in our herd through the feces and urine, so that it seemed certain that the large receiving pen and the food and water receptacles in it were thoroughly contaminated. In

order to demonstrate the communicable nature of this disease under natural conditions, 12 guinea pigs from a different source were obtained and their feces examined. None showed any organisms of the Salmonella group. Autopsies on about 50 animals from the same source showed that the stock was free from Salmonella infection. These 12 animals were placed in the main pen with 42 guinea pigs of the old infected stock. The animals remained together in the one pen from 9-3-38 until 10-24-38 when all survivors were killed. During this time 7 guinea pigs of the old stock died, and 6 of these were found to be infected with *Salmonella typhimurium*. Two of the 12 experimental animals died of this infection, one on 9–24–38 after an illness of 36 hours, the other on 9–29–38 after an illness of 6 days. One experimental animal was found to have the chronic type of Salmonella disease at autopsy on 10-24-38. One other experimental animal (Exp. 10) was found to have the organisms in the feces although cultures of the spleen were negative and there was no gross evidence of infection.

During this experiment, fecal cultures were made from all of the new stock guinea pigs and from about half of the old ones. Table III contains a summary of the results in those cases in which a positive culture was obtained from the feces. It would appear that transitory infection of the animals does occur, probably indicating the occurrence of a carrier state, i.e., the harboring of virulent organisms by an animal not showing any clinical evidence of infection. Of course, the mere presence of bacteria in the feces does not mean that there is actual invasion of the body

TABLE III

*Summary of the Positive Results Obtained in the Cross-Infection Experiment*

| | 9/3 | 9/8 | 9/16 | 9/25 | 10/3 | 10/10 | 10/18 | 10/24 | | |
|---|---|---|---|---|---|---|---|---|---|---|
| G.P. | Feces | Feces | Feces | Feces | Feces | Feces | Feces | Spl. | Feces | Autopsy Findings |
| Exp. 1 | — | | | — | — | — | — | + | — | Nodular spleen |
| Exp. 5 | — | | | + | — | — | — | — | — | Splenic adhesions |
| Exp. 7 | — | | | + | — | — | — | — | — | No lesions |
| | | | | | Died 9/29 | | | | | |
| Exp. 9 | — | | | + | + | | | | | Nodular spleen and liver |
| Exp. 10 | — | | | — | — | — | — | — | + | No lesions |
| | | | | Died 9/24 | | | | | | |
| Exp. 12 | — | | | + | | | | | | Nodular spleen |
| CP 1 | | + | — | — | | — | | — | — | No lesions |
| CP 2 | | + | — | — | | — | | — | — | No lesions |
| CP 10 | | — | — | — | | — | + | + | + | Splenic adhesions |
| CP 12 | | — | + | | | — | | — | — | No lesions |
| | | | Died 9/20 | | | | | | | |
| CP 13 | | — | — | + | | | | | | Septicemia |
| CP 15 | | | — | + | | — | | — | — | No lesions |

Exp. = Guinea pigs from the new, uninfected stock
CP = Guinea pigs from the original, infected herd

tissues. Reference has already been made to an instance (Exp. 10) where the bacteria were apparently living in the intestinal tract without harming the host. Table III shows also that ingestion of the organisms does not usually lead to clinical infection, as only 3 out of 9 of the animals from whose feces *Salmonella typhimurium* was recovered succumbed to the infection. It would seem that the animals either recover fairly promptly or succumb, as we found no instance of prolonged shedding of the bacteria unless the animal was clinically ill.

Raebiger [19] states that agglutinins are formed during the course of the disease and may be used for determining the presence or absence of infection in a herd. From 47 of our animals blood was taken for agglutination tests. Of 4 animals dying of the chronic disease, 2 had no agglutinins while the other 2 had a titer of only 1–10. One animal killed after an illness of almost 2 weeks had a titer of 1–100. Of 4 animals which appeared to be healthy, but were found to be infected at autopsy by culture, 3 had no agglutinins and the 4th a titer of 1–400. The last presented no anatomical lesions although the culture of the spleen showed that *Salmonella typhimurium* was present. Five animals were killed from which no positive cultures were obtained at autopsy; 3 of these had no agglutinins, 1 a titer of 1–400 but no anatomical evidence of past infection, 1 a titer of 1–10 with many fibrinous adhesions about the spleen. Conclusions cannot be drawn from the remaining animals because of insufficient data. The highest titer found was 1–800 in a pregnant female, never known to have been infected and which had no anatomical evidence of past infection at autopsy. Her history before reaching our pens is unknown. From the data gathered it would appear safe to conclude only that an animal may develop agglutinins against the infecting organism, but the absence of such agglutinins does not mitigate against the presence of "pseudo-tuberculosis," either present or past, in the herd.

The original source for this infection is not known although the dealer's own breeding stock was found to be infected. He stated that the guinea pigs on his farm never showed any signs of illness. Those animals examined by us appeared healthy while alive although definite pathological and bacteriological evidence of infection was obtained from them at post-mortem examination. It seems likely that this was a latent infection which was stirred up by the change in diet and the inoculation given the guinea pigs in this laboratory. Such factors are difficult to evaluate, and no control experiment was carried out by us to determine the truth of this hypothesis. Other writers on this subject have found such factors to exist. Edington [8] found that low vitamin diets, severe temperature changes, or mechanical injury to the gastrointestinal tract rendered animals more susceptible to infection. Nelson [14] noted that pregnant sows were most apt to be infected, an observation which was not found to be true among our animals. Okamato [20] found that the parenteral injection of organ extracts was enough to activate a latent infection already present in the guinea pig.

Our efforts in combating this epizoötic were directed toward the prevention of spread of the disease rather than to the cure of the individual animals. All the small cages were cleaned with hot water and sterilized after each lot of animals was killed. The large receiving pens were likewise thoroughly cleaned with hot water and lysol before any new guinea pigs were admitted to the colony. Scattered reports in the literature indicate that fresh vegetables, when fed to the animals in large

amounts, will aid in combating the disease; in our hands the results obtained were unsatisfactory. Vaccines and immune sera were not tried although Howell and Schultz,[5] Nichols and Stimmel,[21] and Rake [22] have reported some success with the use of the former. By the use of simple cleansing methods, our colony has been free of infection for the past 6 months.

## SUMMARY

1. An outbreak of "pseudo-tuberculosis" among guinea pigs is reported.

2. Chronic Salmonella infection of guinea pigs due to *Salmonella typhimurium* cannot be differentiated from tuberculosis by gross anatomical or histological methods. The demonstration of tubercle bacilli in routine smears made from the caseous lesions in the guinea pig is the only reliable way to tell the two conditions apart.

3. The infection is probably spread by means of infected feces and urine. A true carrier state, temporary at least, may develop in individual animals.

4. The organisms are most often found in the spleen. Inability to demonstrate the organisms in fecal cultures does not mean that the animal is not infected.

5. Agglutinins may or may not be formed during the course of the disease. When present they are indicative of successfully combated previous infection rather than of present infection.

6. The presence of a Salmonella infection is not incompatible with an appearance of exceedingly good health on the part of the animal.

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# Zoölogical Problems Relative to Meat Inspection and Their Bearing on Public Health

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OF THE hundreds of species of protozoa, helminths, and arthropods that parasitize animals that are used for human food, comparatively few are of importance in meat inspection. From the viewpoint of livestock production all parasites must be regarded as injurious or potentially injurious. From a meat inspection standpoint, however, the only parasites that are regarded as important are (1) those which occur in the edible portions of carcasses and are directly transmissible to man; (2) those which occur in the edible portions of carcasses and are indirectly transmissible to man through other hosts; and (3) those which, by their mere presence, or because of the pathological conditions which they produce in the edible portions of carcasses, render the affected parts objectionable as human food. Naturally, the parasites that are directly transmissible to man are of greatest importance, and the meat inspection regulations of various countries contain provisions for eliminating this danger to human health.

## PARASITES DIRECTLY TRANSMISSIBLE TO MAN

Fortunately, the only species of parasites occurring in the flesh of food animals that are directly transmissible to man are the bladder worms of the beef and pork tapeworms, respectively, and the larvae of trichina. While tapeworms may be more or less deleterious to human health and may even affect seriously delicate and nervous persons, the pathogenicity of trichinae has been established beyond question, the latter parasites being capable of producing serious illness and even death in human beings. The prevention of human infestation with the three species of parasites mentioned cannot be wholly effected through meat inspection alone; meat inspection can and does assist materially in controlling infestations of human beings with these worms. A system of meat inspection which supervises the slaughter of all food animals, coupled with care in the preparation of meat for the table, should eventually control and even eradicate these three parasites that are directly transmissible from food animals to human beings.

## RECENT INVESTIGATIONS ON CYSTICERCI IN BEEF AND THEIR BEARING ON MEAT INSPECTION

During the last few years scientific investigations regarding the vitality of cysticerci of *Taenia saginata* have led to an important modification in meat inspection procedure with regard to this

parasite. Up to 1931 it was generally assumed that the vitality of cysticerci in beef was completely destroyed following the retention in the cooler of infested carcasses for a period of 21 days. This assumption was based on tests first carried out by Perroncito [4] in Italy, and later extended greatly by Ostertag [3] in Germany. Perroncito determined that all cysticerci in an infested calf carcass were dead 14 days after the animal had been slaughtered; Ostertag showed, however, that the natural death of cysticerci in beef did not occur uniformly in a 2 week period, but that in 3 weeks following the death of the infested host animal all the cysticerci were dead. Ostertag's observations were based on various tests applied to determine the vitality of the cysticerci following the retention of the infested carcass for 3 weeks, these tests being supplemented by human feeding experiments in which Ostertag himself and a number of assistants and students of the Berlin Veterinary College participated. These tests involved the swallowing by the human volunteers of cysticerci obtained from beef carcasses which had been retained for 21 days, with negative results so far as the development of tapeworms in the human intestine was concerned. On the basis of this searching investigation, the retention for 21 days of beef carcasses infested with cysticerci was incorporated in the meat inspection regulations of Germany and was later incorporated in the regulations governing the meat inspection of the U. S. Department of Agriculture.

In 1931 Schmey and Bugge in Germany published [8, 9, 10] the results of investigations which cast considerable doubt on the effectiveness of a 21 day retention period in completely destroying the vitality of *Cysticercus bovis*. These investigators determined that not all of the cysticerci present in beef were destroyed in 21 days; some of the cysticerci present in lightly infested beef carcasses still showed some sign of life even after 41 days' retention, viability being determined by certain biological tests involving the demonstration of the ability of the cysticerci to evaginate the heads and the demonstration of ciliary movement in the flame cells of these worms. Schmey and Bugge determined, moreover, the important fact that cysticerci removed from carcasses that had been retained by them in the cooler for 28 days were still capable of developing into strobilate tapeworms in the intestine of man. Of the 6 volunteers who swallowed a total of 105 cysticerci from beef that had been retained 28 days, 3 became infested and 6 mature strobilate tapeworms were removed from these subjects following anthelmintic medication.

The work of these investigators having been confirmed by Clarenburg [1] in Holland, so far as the viability of cysticerci was concerned following the retention of infested carcasses up to 41 days, investigations were undertaken in the Zoölogical Division of the Bureau of Animal Industry to test the soundness of these new observations. In 1933, 3 infested beef carcasses were retained in the cooler of a meat packing establishment in Chicago for 21 days or longer; at the end of 21 days it was determined that most of the cysticerci showed movement and that from 60 to 100 per cent of various lots of these worms were capable of extruding their heads in suitable media, this being sound evidence of viability. Nearly similar results were obtained when infested carcasses were retained in the cooler for 25 and 26 days, respectively, but after retention for periods ranging from 31 to 48 days there was no evidence of life in the cysticerci. One worker in the Zoölogical Division swallowed several cysticerci from beef that had been retained in the cooler for 26 days; although these cysticerci were

still showing signs of life, he failed to show evidence of infestation with tapeworms.

Although the evidence obtained in our investigations was at variance with those published abroad, the results obtained did show that the 21 day retention period, previously accepted by parasitologists and meat inspection experts as adequate for the destruction of the vitality of beef cysticerci, did not destroy life in those parasites in all cases.

The discrepancy in the results obtained by Ostertag many years ago and those obtained more recently in Germany have been ascribed to the improvement in refrigeration in recent years, the assumption being that the superior cooler refrigeration facilities at the present time favor the survival of cysticerci rather than hasten their devitalization. According to this assumption, the imperfect cooler refrigeration of previous years was responsible for the comparatively early death of cysticerci by hastening certain changes in the meat that produced deleterious effects on the parasites. It is quite possible that the differences between our results and those in Europe might be accounted for, at least in part, on the basis of differences in conditions prevailing in the meat coolers used in this country and those used abroad. Regardless of the true explanation, however, our work demonstrated conclusively that it was no longer proper to include in the regulations the 21 day retention period as an effective method for the destruction of the vitality of *Cysticercus bovis*, and this requirement was accordingly eliminated from the regulations. The alternative requirement, involving the refrigeration of infested carcasses for not less than 6 days at a temperature not higher than 15° F., based on investigations by the late Dr. B. H. Ransom [6] has been retained in our regulations because the more recent investigations in Europe on the effects of low temperatures on the vitality of cysticerci have shown that the retention period required under federal meat inspection is more than adequate to destroy life effectively in these parasites. According to the later European work by Schmey and Bugge,[8] *Cysticercus bovis* loses its vitality when infested carcasses are retained for 4 days at a temperature of 14° to 17.6° F. (—10° to —8° C.) the effective period being 2 days less than that required under our regulations. Viljoen [12] has concluded also that no cysticerci will remain viable after 6 days' continuous freezing.

### INVESTIGATIONS ON TRICHINAE

During the last 10 years human trichinosis has been assuming increasing importance as a public health problem; the number of cases reported in the medical literature and elsewhere during this time has shown a sharp increase. From 1842, when trichinosis in man was first diagnosed in this country, up to 1915, when all the known cases were summarized by Ransom,[7] the total number of recorded cases of human trichinosis in this country was approximately 1,500. From 1915 to about 1925 the number recorded annually in this country was comparatively low. Since 1925 there has been a marked increase, the total number in 1937 being between 5,000 and 6,000. In other words, the number of cases during these 22 years is from 3 to 4 times that recorded for the previous 73 years.

While it cannot be determined on the basis of available evidence whether the increase in the cases of human trichinosis in this country is only apparent or real, in the opinion of the writer this increase is largely apparent because of the increased interest in this disease by the medical profession, and their increasing vigilance in establishing

a correct diagnosis. The increased interest by the medical profession is due almost entirely to certain statistical information on the prevalence of trichinae in human diaphragms examined by various investigators by the digestion or other improved technic.

Following Queen's report [5] which showed that trichinae were found by him in about 17 per cent of 500 diaphragms obtained from consecutive human necropsies in Rochester, N. Y., and in about 27 per cent of 200 diaphragms from Boston, a number of workers who have followed up this type of investigation have brought to light facts which show conclusively that the incidence of infestation of man with trichinae in the United States is much greater than had ever been suspected. In a recent summary covering 1,000 diaphragms examined at the National Institute of Health, Washington, D. C., Nolan and Bozicevich,[2] the incidence was found to be 17 per cent.

It is important to bear in mind that the surprisingly high incidence of trichina infestation in human beings in the United States, as determined by the examination of diaphragms by the digestion technic or by sectioning, or by repeated examination of press preparations, is of significance, despite the fact that very few of the individuals in whose diaphragms trichinae were found post-mortem were known to have suffered from trichinosis during life. The findings indicate conclusively that many persons intentionally or unwittingly eat pork that is raw or nearly raw, and as a result acquire an infestation with trichinae. This in turn indicates that sources of trichina infestation are present in the United States and that the extent of this infestation is sufficient to affect in some localities up to 25 per cent or more of the population, assuming that the sampling of human diaphragms, as recorded, was adequate from a statistical viewpoint.

INCIDENCE OF TRICHINAE IN SWINE

In order to ascertain the prevalence of trichinae in swine, and to determine the main sources of this infestation, so far as human beings are concerned, the Zoölogical Division in coöperation with the Meat Inspection Division of the Bureau of Animal Industry inaugurated in 1933 an extensive survey, the object being to determine by random sampling the incidence of trichinae in garbage-fed hogs and in grain-fed hogs in various parts of the United States, to check the effectiveness of the Bureau's prescribed methods for destroying the vitality of trichinae in meat food products containing pork muscle tissue that are customarily eaten without cooking, and incidentally to ascertain the prevalence of trichinae in prepared meat food products of kinds that are not processed for the destruction of life in trichinae under the requirement of the U. S. Bureau of Animal Industry. The latter served as a check on the technic used in our investigations for the processed products. The technic used involved the artificial digestion in an incubator of approximately ½ lb. samples; this was also followed for samples of fresh pork.

The results in grain-fed and in garbage-fed hogs to June 30, 1937, have been summarized by the writer,[11] as follows:

Out of 6,622 * samples of diaphragm muscle tissue obtained from as many grain-fed hogs originating in the South, the Middle West, the North-Central States and in some of the Eastern States, only 60, or 0.91 per cent, were found to be infested with trichinae; out of 6,484 * diaphragm samples from as many garbage-fed hogs, 286, or 4.41 per cent, were found to be infested. These data show an incidence of trichinae in garbage-fed hogs about 5 times as great as in grain-fed hogs.

* The figure given in the previous publication is incorrect due to a typographical error.

Since July 1, 1937, additional data have been collected, with results that are even more striking. Out of 2,901 diaphragms collected in federally inspected establishments in Chicago and Kansas City from grain-fed hogs originating in the Middle West, 34, or 1.17 per cent, contained trichinae; the number of trichinae in individual diaphragms ranged from 1 to 1,113. However, of the positive diaphragms 76.4 per cent contained less than 5 larvae, this type of infestation being practically negligible. Out of 2,847 samples of diaphragms obtained from as many garbage-fed hogs in an important garbage-feeding center on the Atlantic seaboard, 286, or 10.04 per cent, contained trichinae; the number of trichinae in individual diaphragms ranged from 1 to 69,700. Of the positive diaphragms only 19.3 per cent contained less than 5 larvae.

These data demonstrate conclusively that pork from garbage-fed hogs is the main source of trichinosis for man, if such pork is eaten raw or after only partial cooking or inadequate curing. The available data are sufficiently extensive to warrant the preliminary conclusion that about 1 per cent of the grain-fed hogs in this country harbor trichinae, whereas from 4.5 to 10 per cent of garbage-fed hogs are infested, the degree of infestation being much greater than in hogs not fed on garbage.

### INCIDENCE OF TRICHINAE IN MEAT FOOD PRODUCTS CONTAINING PORK MUSCLE TISSUE

During the last 5 years there have been examined in the laboratories of the Zoölogical Division 10,754 individual ½ lb. samples of various meat food products containing pork muscle tissue. The samples were obtained through federal inspectors in charge of meat inspection, who submitted, in connection with each sample, data as to the method of processing and information as to whether the product was of a kind customarily eaten without cooking in the home. Each sample, properly identified as to class and place of origin, was ground and digested in a fluid containing pepsin and hydrochloric acid in an incubator for about 24 hours. Live or dead trichinae, if present, could be recovered from the sediment of the digestive fluid.

Of the samples so examined, 9,788 were from products processed under Bureau requirements to insure the destruction of life in trichinae, and 966 were from products not so processed.

The samples of products processed for the destruction of life in trichinae—such as bologna, frankfurters and Vienna sausage, various kinds of dry or summer sausage, cooked ham and Italian-style ham—were obtained from federally inspected establishments and represented a good cross-section of these products that are released to the consuming public. The processing included heating and special refrigeration, as well as the various prescribed curing methods. The results obtained were as follows:

Out of the 9,788 specially processed samples, trichinae were found in only 75; 71 contained only dead, uncoiled trichinae and 3 contained trichinae showing feeble movements. Feeble trichinae were found in 3 samples of smoked sausage, the number in the 3 samples being 1, 1, and 3, respectively; a single coiled but dead trichina was found in a skinless frankfurter. It is important to note that the feeble worms referred to were not tightly coiled and deep brown in color as are decapsuled trichinae whose vitality has not been reduced by heat, cold, or other processing. They were loosely coiled and rather pale, but slight movements could be induced by the application of a heated glass rod to the bottom of the glass dish in which they were. Large num-

bers of worms similar in appearance and activity to those just described have been fed to rats in our laboratory at various times in order to determine whether they are capable of surviving and growing in the intestine and producing a new generation of worms. In no instance have such worms succeeded in remaining alive in the intestine of susceptible rats, as determined by the results of a searching examination of the intestines of these experimental hosts 24 hours later. A number of rats to which such worms were fed by stomach tube, or which swallowed relatively large quantities of meat containing such worms, were killed after one month. Following the removal of skin and viscera, the entire carcass of each rat was digested without yielding any trichinae.

Of the 966 products not processed under the requirements as to the destruction of the vitality of trichinae, 480 (nearly 50 per cent) contained these parasites; of the 480 positives, 435 contained only dead trichinae, and the remaining 45 contained live and dead trichinae, the number of live worms per sample ranging from 1 to 14.

### ECHINOCOCCUS CYSTS

Of the parasites occurring in the edible parts of food animals that are transmissible to man indirectly, that is, through an intermediate host, hydatid cysts, *Echinococcus,* are the most important. The cystic stage of this parasite develops into strobilate tapeworms in the intestines of dogs, cats, and certain wild carnivores, the feces of the infested carnivores constituting a source of infection for human beings and all classes of domestic animals. An infestation with hydatids in man is a serious condition, and treatment involves a major surgical operation for the removal of the cysts. As the cysts usually lodge in the liver and may occur in other vital organs, particularly the lungs, it is evident that their removal by surgery involves a serious risk to life.

The occurrence and suspected occurrence of *Echinococcus* infestation in man in this country may be inferred from the repeated requests which come to the Bureau of Animal Industry for an antigen that is used in the complement-fixation test for the diagnosis of this parasitic infestation in man. Through an arrangement with the U. S. Naval Medical School, swine livers infested with *Echinococcus* are forwarded to that school from time to time for the preparation of the antigen and its distribution to physicians and hospitals. During the last 3 or 4 years it has been rather difficult to obtain infested livers from federal inspectors. The station at Richmond, Va., for years a reliable source of supply, is now having some difficulty in obtaining the material needed.

It is probable that meat inspection is largely responsible for what appears to be a sharp decrease in the incidence of *Echinococcus* infestation in swine and other animals. The condemnation of affected organs and cysts and their consignment to the tank cuts down the source from which dogs, cats, and wild carnivores acquire the hydatid tapeworm, thereby destroying the vicious cycle of a parasite that is dangerous to human and animal health.

### SUMMARY

An effective system of meat inspection promotes public health (1) through the condemnation of parts of or entire carcasses of food animals containing parasites that are transmissible to man directly and indirectly; (2) through special processing for the destruction of trichinae, and of cysticerci not discoverable by macroscopic examination; and (3) through the condemnation of parts of carcasses or of entire carcasses in which parasitic infestations not trans-

missible to man, but objectionable from esthetic and other standpoints, are so generalized that the parasites and lesions cannot be removed by trimming.

Meat inspection is also an effective control measure for certain parasites, notably tapeworms, that occur as immature forms in edible portions of carcasses; through the condemnation of parts of carcasses so affected, the vicious cycle of these parasites is interrupted. Meat inspection must be regarded, therefore, as one of the important prophylactic measures for the control of parasites affecting livestock as well as human beings.

Certain parasitic infestations transmissible from food animals to human beings cannot be controlled by meat inspection alone; however, a sound inspection system can and does contribute materially to control of certain parasitic infestations of man; in the absence of inspection these parasites would probably constitute a serious human health problem.

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# Meningococcus Meningitis in the District of Columbia

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IN general the occurrence of meningococcus meningitis in the District of Columbia has been similar to that in other urban communities of the United States.[1, 2, 3] Since 1910 there have been two sharp outbreaks of the disease. The epidemic in 1918 was preceded by a moderate increase in prevalence in 1917. The 1935 outbreak was more explosive in type and during the following 2 years the incidence was above the normal expectancy (see Figure I). In 1931 the incidence of the disease was above the average but not severe enough to classify as an epidemic. In the intervals between these outbreaks the incidence has been at a fairly low endemic level.

*Seasonal occurrence*—As in other parts of this country, the disease has been reported most frequently in the late winter and spring months, and least frequently in the fall. The epidemic in 1935 had its peak in March. This epidemic was characterized by a rapid increase in cases from January to the latter part of March when the peak was reached (see Figure II). Most of the cases reported early in the epidemic came from well defined areas in the city where housing and sanitary conditions have been far from desirable.

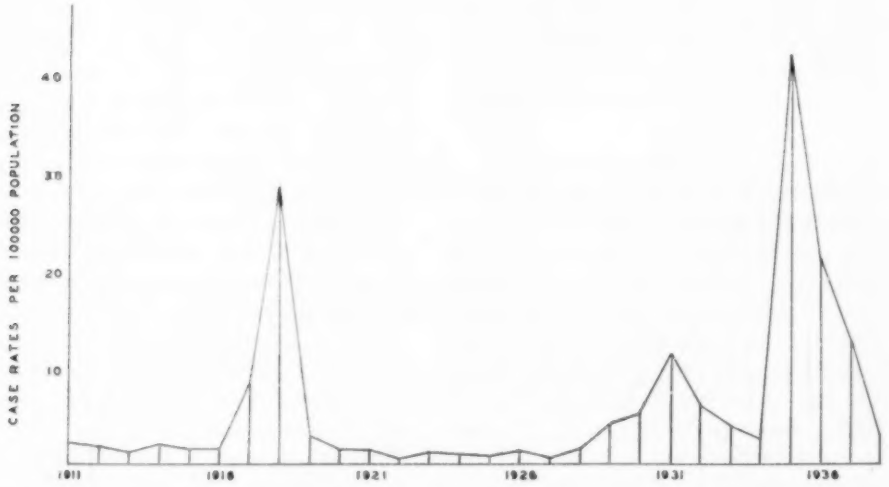
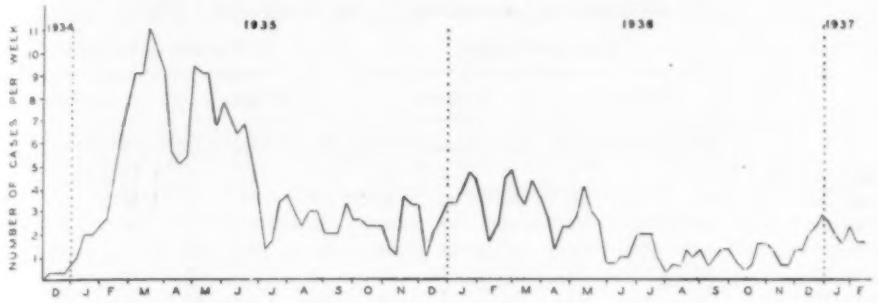



FIGURE I—Incidence of Meningococcus Meningitis in the District of Columbia, 1911–1938

FIGURE II—Number of Cases of Meningococcus Meningitis Reported in the District of Columbia by Weeks from December, 1934, to February, 1937, inclusive




The secondary rise in the number of cases in May of 1935 was due to a large number of cases among colored persons reported from all sections of the city. The decline in the number of cases reported by weeks was much more gradual than the increase at the beginning of the outbreak. Throughout 1935 the number of cases reported each week continued at a fairly high level. The seasonal increase in cases in the winter and spring of 1936 was a gradual one and to a point far below the peak of the previous year. Throughout 1936 the number of cases continued to decrease. The seasonal increase in cases in 1937 was still lower than in 1936, and in 1938 the disease once more assumed a low endemic level.

*Racial distribution*—As shown in Table I, case rates and death rates from meningococcus meningitis in the District of Columbia have been higher in the colored population than in the white, with few exceptions. The ratio of colored to white rates has usually been about 2 or 3 to 1. In some years the incidence of the disease has been about equal in the two racial groups, but in years of epidemic or increased prevalence the colored rates have been 2 or 3 times higher than the white.

*Sex differences in incidence and mortality*—The incidence of the disease in males has invariably been higher than in females. The ratio of male to female cases has been about 2 to 1 in both white and colored persons. Under 20 years of age the male to female ratio was about 1.5 to 1, and from 20 to 40 years varied from 3.1 to 6.1.

TABLE I

*Number of Cases and Deaths, Rates per 100,000 Population, and Case Fatality per 100 Cases, from Meningococcus Meningitis, 1935–1937*

| | Cases | | Deaths | | Case Rate | | Death Rate | | Case Fatality Rate | |
|---|---|---|---|---|---|---|---|---|---|---|
| Year | White | Colored | White | Colored | White | Colored | White | Colored | White | Colored |
| 1928 | 5 | 2 | 3 | 1 | 1.4 | 1.7 | 0.9 | 0.8 | 60.0 | 50.0 |
| 1929 | 6 | 10 | 2 | 8 | 1.7 | 7.6 | 0.6 | 6.1 | 33.3 | 80.0 |
| 1930 | 12 | 8 | 5 | 3 | 3.4 | 6.0 | 1.4 | 2.2 | 41.6 | 37.5 |
| 1931 | 26 | 25 | 11 | 15 | 7.1 | 18.1 | 3.0 | 10.8 | 42.2 | 60.0 |
| 1932 | 16 | 19 | 9 | 2 | 4.2 | 13.2 | 2.3 | 1.4 | 56.1 | 10.5 |
| 1933 | 12 | 12 | 7 | 1 | 3.0 | 8.0 | 1.8 | 0.7 | 58.5 | 8.3 |
| 1934 | 7 | 2 | 0 | 2 | 1.7 | 1.3 | 0 | 1.3 | 0 | 100.0 |
| 1935 | 93 | 136 | 39 | 47 | 22.0 | 85.6 | 9.1 | 29.3 | 42.0 | 34.6 |
| 1936 | 52 | 57 | 24 | 35 | 11.8 | 34.4 | 5.4 | 19.9 | 46.1 | 61.4 |
| 1937 | 30 | 60 | 15 | 12 | 6.6 | 15.2 | 3.3 | 7.0 | 50.0 | 46.0 |

TABLE II

*Number of Cases and Deaths, Ratio of Male to Female Cases, and Case Fatality Rates, from Meningococcus Meningitis, by Age Groups, 1935–1936*

| | Number of Cases | | | | | | Number of Deaths | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | White | | | Colored | | | White | | | Colored | | |
| | Male | Female | Total | Male | Female | Total | Male | Female | Total | Male | Female | Total |
| Under 1 year | 1 | 2 | 3 | 6 | 4 | 10 | 0 | 1 | 1 | 3 | 4 | 7 |
| 1 to 4 | 13 | 7 | 20 | 26 | 19 | 44 | 3 | 1 | 4 | 4 | 6 | 10 |
| 5 to 9 | 5 | 3 | 8 | 15 | 14 | 29 | 1 | 1 | 2 | 8 | 2 | 10 |
| 10 to 19 | 18 | 13 | 31 | 37 | 11 | 48 | 11 | 3 | 14 | 11 | 6 | 13 |
| 20 to 29 | 28 | 7 | 35 | 22 | 7 | 29 | 9 | 2 | 11 | 10 | 4 | 14 |
| 30 to 39 | 19 | 3 | 22 | 13 | 5 | 18 | 11 | 2 | 13 | 10 | 3 | 13 |
| 40 to 49 | 12 | 3 | 15 | 2 | 4 | 6 | 8 | 2 | 10 | 2 | 4 | 6 |
| 50 and over | 5 | 5 | 10 | 4 | 4 | 8 | 3 | 5 | 8 | 3 | 2 | 5 |
| Unknown | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| All ages | 102 | 43 | 145 | 125 | 68 | 193 | 46 | 17 | 63 | 51 | 31 | 82 |

| | Case Fatality Rates | | | | Ratio Male to Female Cases | |
|---|---|---|---|---|---|---|
| | White | | Colored | | | |
| | Male | Female | Male | Female | White | Colored |
| Under 1 year | 0 | 50.0 | 50.0 | 100.0 | 0.5 | 1.5 |
| 1 to 4 | 23.1 | 14.3 | 15.4 | 33.3 | 1.9 | 1.4 |
| 5 to 9 | 20.0 | 33.3 | 53.3 | 14.3 | 1.7 | 1.1 |
| 10 to 19 | 61.1 | 23.1 | 30.0 | 55.5 | 1.4 | 3.3 |
| 20 to 29 | 32.1 | 28.5 | 45.4 | 55.5 | 4.0 | 3.1 |
| 30 to 39 | 57.9 | 66.6 | 77.0 | 60.0 | 6.3 | 2.6 |
| 40 to 49 | 66.6 | 66.6 | 100.0 | 100.0 | 4.0 | 0.5 |
| 50 and over | 60.0 | 100.0 | 75.0 | 50.0 | 1.0 | 1.0 |
| All ages | 45.1 | 39.5 | 40.8 | 45,6 | 2.4 | 1.9 |

*Case fatality rates*—It can be seen in Table I that case fatality rates have varied from year to year, with less variation for white than colored cases. The average for the 10 year period from 1928 to 1937 was 44 per cent for both white and colored groups. Although the incidence of the disease has been higher among males, the fatality rates have been approximately the same for males and females. For the cases reported in 1935 and 1936 the case fatality for white males was 45.1 per cent, 39.5 for white females, 40.8 for colored males, and 45.6 per cent for colored females.

*Age distribution of cases and deaths*—The distribution of cases showed wide variations in the different age groups, the greater proportion of cases being in the younger groups. Forty-two per cent of white cases and 68 per cent of the colored occurred in persons under 20 years of age. The case rates per 100,000 population for the various age groups likewise reflect the greater prevalence in the younger age groups.

There was very little difference in the death rates from meningococcus meningitis by age groups in the white population, but in the colored the rates were much higher in children under 5 years of age. In the latter there was a decline in mortality as age increased.

Case fatality rates were fairly low in the younger age groups and increased

with age. Case fatality was higher in infants under 1 year of age than in the 1 to 4, and 5 to 9 year groups, but appreciably lower than in the older ages, *i.e.*, 40 years and over. The fatality rates for males and females were essentially the same when all ages or specific age groups were compared.

*Geographical distribution in the District of Columbia*—The geographical distribution of meningococcus meningitis cases in the District of Columbia has exhibited no unusual characteristics. In general the disease has occurred most frequently where housing conditions are bad and poor hygiene has been most commonly practised. In the white population the incidence in 1935 and 1936 was highest in the central part of the city where a large proportion of the population has lived under these undesirable conditions. In this area a group of 7 census tracts, in which about 8 per cent of the white population of the city resides, furnished 28 per cent of the white cases in 1935 and 1936. The case rate for this group was 57 per 100,000 population, and for the remainder of the city's white population the rate was 12.6. However, in other scattered sections where environmental conditions were about as unsatisfactory, the case rates were about the same as those of the group described above. The incidence was generally low (10 or less per 100,000 population) in the better residential sections.

The disease was more evenly distributed in the colored population.

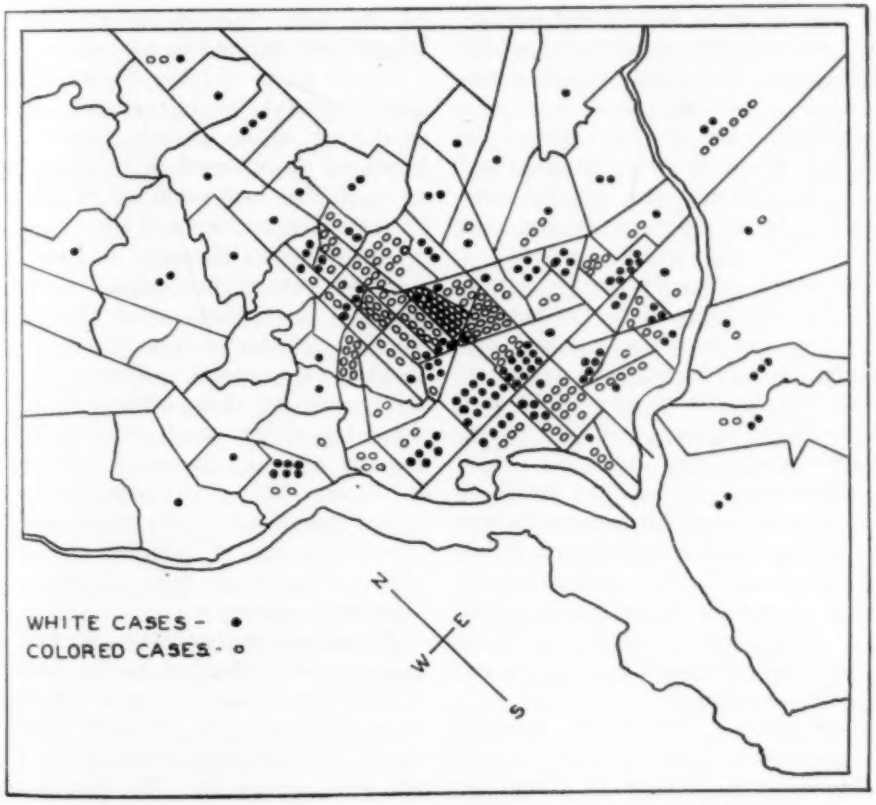



FIGURE III—Geographical Location of Cases of Meningococcus Meningitis Reported in the District of Columbia during 1935 and 1936

Sixty per cent of all colored cases reported in 1935 and 1936 came from a section roughly 12 blocks wide and 30 blocks long extending through the center of the city immediately west of North Capitol and South Capitol Streets. Fifty per cent of the colored population in the District of Columbia resides in this section. The case rate was 70 per 100,000 population. Other smaller areas in the city where there was a concentration of colored population had approximately the same incidence rate as the larger area. The more even distribution of the disease in the colored population probably occurred because a large proportion of these people have lived under conditions which have favored the spread of the disease. It should be noted that there was not a wide difference in case rates of white and colored populations, 57 and 70, respectively, which lived under somewhat similar conditions.

The first cases to be reported in 1935 came from the two areas described above and the majority of the cases reported from other areas occurred later in 1935 and 1936.

*Occurrence in family groups, institutions, etc.*—Multiple or second cases in family groups were not reported frequently. In 1935 and 1936, two cases in the same family occurred only once among white persons, and in two instances among the colored. Four cases occurred in one colored family.

There were numerous instances where two or three cases were reported within a short space of time from adjoining houses or in the same block. Two cases were reported from the same address but in different families on two different occasions.

Meningococcus meningitis occurred in a number of institutions in or controlled by the District of Columbia. Four cases were reported from three of the penal institutions in 1935 and 1936. Eleven cases occurred among transients early in 1935. These men were housed in two rooming houses patronized exclusively by transients. In 1936 one case occurred in the National Training School for Boys, and in 1937 there were 6 cases in this institution. The average enrollment is about 400 boys, mostly between 15 and 20 years of age.

*Study of hospital admissions*—Practically all of the cases of meningococcus meningitis reported in the District of Columbia have been admitted to the various hospitals in the city. Hospital records of 118 white and 167 colored patients with the disease who were admitted to two hospitals, were reviewed for information of epidemiological interest. This group of cases, admitted during 1935, 1936, and 1937, constituted a little more than two-thirds of all the cases reported to the Health Department during this period.

In this series of cases studied, it appears that fatality rates depend more on the age of the patients than on the length of the interval between the onset and the institution of treatment. Patients whose treatment began 2 days or less following the onset had as high a fatality rate as those whose treatment began 3 to 5 days after the onset. Further analysis of the former group showed no essential difference in fatality rates of those admitted on the same day as the onset, those admitted the day after, or the second day after the onset. This group undoubtedly included nearly all of the severe or fulminating types of infection many of which succumb in spite of early and vigorous treatment.

A comparatively small number of patients were admitted to the hospital 6 to 9 and 10 days or more after the onset of symptoms. Fatality rates in the 6 to 9 day group were higher than all other groups but the number was too small to permit drawing any definite conclusions. Those admitted

TABLE III

*Fatality Rates per 100 Cases of Meningococcus Meningitis Admitted to Two Hospitals in 1935 to 1937, Inclusive, by Age Groups and Number of Days After Onset of Symptoms*

| Days After Onset | Age Groups | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Under 1 Yr. | 1–4 | 5–9 | 10–19 | 20–29 | 30–39 | 40–49 | 50+ | Total |
| | | | | *White* | | | | | |
| 2 days and under | 33.3 | 9.1 | 37.5 | 35.3 | 26.6 | 66.6 | 50.0 | 66.6 | 34.3 |
| 3 to 5 days | 33.3 | 0 | 0 | 22.2 | 16.6 | 33.3 | 33.3 | 80.0 | 31.2 |
| 6 to 9 days | 100.0 | 0 | x | x | 0 | 100.0 | x | 33.3 | 50.0 |
| 10 days and over | x | x | x | x | 25.0 | 33.3 | 30.5 | x | 25.0 |
| Total | 42.8 | 7.1 | 33.3 | 30.8 | 25.0 | 60.0 | 37.5 | 63.3 | 34.2 |
| | | | | *Colored* | | | | | |
| 2 days and under | 33.3 | 13.0 | 30.0 | 30.8 | 53.8 | 71.4 | 100.0 | 100.0 | 37.3 |
| 3 to 5 days | 33.3 | 9.1 | 0 | 38.1 | 43.0 | 55.0 | x | 0 | 32.0 |
| 6 to 9 days | 100.0 | 50.0 | 33.0 | 100.0 | 100.0 | 50.0 | x | x | 63.6 |
| 10 days and over | 100.0 | 0 | 0 | 0 | x | x | 100.0 | x | 28.5 |
| Total | 42.8 | 13.0 | 28.0 | 35.7 | 50.0 | 62.5 | 100.0 | 80.0 | 38.3 |

x No Cases

on the 10th day or later had the lowest fatality rate.

It is interesting to note that fatality rates increased materially with age whether the patients were admitted early or late following the onset of symptoms.

Definite information on the different types of meningococci present in the spinal fluid or blood stream of the hospitalized cases was available for only a few cases. However, the organism was found by direct smear or by culture in about 85 per cent of all cases. The importance of typing has been pointed out by Branham.[4]

Although the type of treatment varied in the two hospitals, there was no significant difference in case fatality rates among cases of similar age. One hospital admits only children, and the other is an isolation unit of a general hospital. Antitoxin was used extensively in the former and antimeningococcus serum in the latter. In both institutions sulfanilamide or one of its derivatives was used in the treatment of a small proportion of cases in 1937. Case fatality rates in antitoxin treated and antimeningococcus serum treated cases were essentially the same when cases of the same age were compared. The series of cases in which sulfanilamide therapy was used was too small to warrant drawing any conclusions regarding its effectiveness.

*Discussion*—In general the epidemiological features of meningococcus meningitis have been no different in the District of Columbia than in other large cities. There have been epidemics at irregular intervals, a greater incidence in late winter and spring, a greater incidence in colored persons, a relatively high ratio of male to female cases, and a higher case fatality in older than in young persons. The relatively high incidence and mortality rates among males has been due mainly to the great excess of cases in males from 20 to 40 years of age. Differences in occupation and other environmental factors leading to more frequent exposure to infection have been suggested to account for the greater incidence in males. The fact that case fatality rates

are essentially the same for groups of males and females of the same age lends weight to this suggestion.

Studies in which the effect of various therapeutic agents, particularly sulfanilamide, on the fatality rate of meningococcus meningitis have been reported, were reviewed by the author. Few reports seem to indicate that careful thought has been given certain factors, other than the form of treatment, which affect fatality rates. These factors are: (1) age distribution of cases; (2) type of organism found; and (3) the period of time in which the cases occurred. In order that valid conclusions may be drawn, it is essential that the age distribution of specially treated and control groups be the same, or a sufficient number of cases in various age groups must be included in each series. The classification of cases according to type of meningococcus found is also essential in appraising the effects of various forms of treatment. Series of cases must extend over the same period of time because of the possibility of differences in virulence of the organism from year to year.

Banks [5] in his report on sulfanilamide treatment of meningococcus meningitis took the above mentioned factors into consideration and concluded that sulfanilamide was an effective therapeutic agent. Other reports [6, 7, 8] are less convincing because there are no definite data with respect to factors other than treatment. The superiority of sulfanilamide over other forms of specific therapy does not seem to have been demonstrated satisfactorily up to the present time.

NOTE: The author wishes to take this opportunity of thanking Dr. Sara E. Branham of the National Institute of Health for helpful suggestions in the preparation of this manuscript.

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# A Permanent Nephelometer from Pyrex Glass

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THE use of nephelometry in the standardization of bacterial suspensions has been greatly handicapped because of the instability of the standards usually employed. Standards made with the actual organisms rapidly undergo autolysis even in the presence of strong bacteriostatic agents, and those prepared by chemical precipitation methods have the disadvantage that the crystals grow by accretion and one is soon reluctant to depend upon them. Barium sulphate standards [1] and the usual silica standards [2] have the additional disadvantage that when the proper turbidity is reached transmitted light assumes a greyish-white opalescence, whereas, the bacterial suspension has an amber cast. This discrepancy is due for the most part to the difference in size of the particles.

A method for reducing pyrex glass chips to the size of bacteria and grading them for nephelometric work was discovered entirely by accident while preparing glass chips for grinding rabbit brains for rabies vaccine. Discarded pyrex beakers and flasks were broken into chips about one-half to one inch in size and were placed in a pyrex flask or wide mouth bottle to about one-fourth of the capacity. They were then covered with distilled water and placed on a shaking machine. This machine was run for 45 minutes over a 4″ arc at 120 strokes per minute in order to remove the rough edges from the chips. For lack of sink space the distilled water from these chips was decanted into a cylindrical discard jar. After several days it was noted that the turbid liquid had settled into several distinct layers, one of which closely approached the appearance of typhoid vaccine. Noting this, we became interested in preparing a set of vaccine standards which would not settle out as rapidly as the freshly prepared barium sulphate standards which we had been using. The turbid layer of the glass suspension which most closely resembled the typhoid vaccine was siphoned off and concentrated by evaporation in order to make a range of standards. The glass standards were then calibrated by preparing bacterial suspensions of known count (Helber cell, Wright and Hopkins tube methods) and matching the concentrated solution by dilution to the bacterial standards. The resulting glass standards could not be differentiated from the bacterial suspensions whether in transmitted or reflected light. When the particles were measured it was found that they varied from 0.5 to 3.5

microns in diameter, which is the range of most of the organisms in which we were interested.

The difference in the size of these glass particles and freshly prepared barium sulphate is readily shown if one filters these suspensions through a very hard filter paper. The barium sulphate particles are removed by this filtration but the glass suspension remains turbid. This filtration method has been used by one of us to grade the size of the particles, rather than the sedimentation or centrifugation methods.

The rate of settling of the glass standards is much slower than that of the usual silica or barium sulphate type. Pyrex standards, properly prepared by this technic, will not settle out completely within 3 weeks although signs of sedimentation will be noted within 24 hours. They can be resuspended immediately by shaking a few times and will show no signs of accretion. The chemically precipitated standards will settle out completely within a few hours. Microscopic examination of the glass standard reveals "Brownian movement" similar to that noted in suspensions of bacteria of comparable size and density.

The accuracy of these standards will be seen in Table I.

This accuracy with a nephelometric method is possible only when using standards which match the suspensions in transmitted and reflected light. It is therefore necessary that the size of the particles in the standard be within the size range of the bacteria in the suspension.

TABLE I

*Comparative Counts With Typhoid Vaccine*

| *Stock* | *Nephelometer Reading* | *Direct Count With Levy Hausser Counting Chamber* |
|---|---|---|
| 1 T * | 21,000 M | 20,600 M |
| 2 T | 23,000 M | 22,000 M |
| 3 T | 25,000 M | 24,100 M |
| 4 T | 25,000 M | 26,000 M |
| 5 T | 26,000 M | 25,200 M |
| 6 T | 18,000 M | 15,000 M |
| 7 T | 23,000 M | 20,300 M |
| 8 T | 23,000 M | 22,000 M |
| 9 T | 29,000 M | 27,600 M |
| 1 A † | 24,000 M | 23,400 M |
| 2 A | 27,000 M | 26,700 M |
| 3 A | 27,000 M | 29,000 M |

* Stock T is a combination of 6 strains of *Eberthella typhi.*

† Stock A was made from the Kessel stain No. 501 A.

The stability of these pyrex glass standards is shown by the fact that standards which have been in constant use for 5 years give the same reading now as when originally made. The glass particles measure the same and show no tendency to clump, cement or dissolve. Because of the fact that pyrex glass is not affected thermally or chemically by any of the methods commonly employed in sterilization, these standards can be sealed in ampules and autoclaved to prevent mold or bacterial growth which would interfere with their accuracy.

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# The Occurrence of Atmospheric Lead Resulting from Use of Colored Blackboard Crayons

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THE Wisconsin State Board of Health was recently requested to analyze two samples of yellow blackboard crayons suspected of containing lead. The analysis showed they contained respectively 7.0 and 8.8 per cent lead. Subsequent discussions with school officials disclosed that yellow blackboard crayons were extensively used, especially in defective vision classes. The present tendency of substituting light colored writing surfaces for blackboard necessitates the exclusive use of colored crayons. The possibility of lead poisoning due to inhalation of lead dust produced while using some of the colored crayons became apparent and the problem seemed sufficiently important to warrant investigation. The investigation was divided into 3 portions, (a) evaluation of the lead content of crayons, (b) determination of the amount of crayon used under various classroom conditions, and (c) amount of lead present in the air when crayons containing lead pigment were used.

Twenty-seven crayons representing 6 different manufacturers were analyzed for their lead content. The crayons were obtained from various schools or school supply houses in Wisconsin without selection. Lead was determined by electrolytic separation, interfering metals being eliminated in the procedure. The analytical results are given in Table I. Of the samples analyzed, lead was found only in the yellows, oranges, and greens, the quantity varying between 1.5 and 12.0 per cent by weight. These results are in accord with those reported by Jephcott.[1] One yellow crayon was submitted for analysis which contained no lead pigment, and information has been obtained that another manufacturer is developing a lead-free yellow crayon.

## RATE OF CRAYON USAGE

Studies made in actual classrooms showed that the amount of crayon used varies considerably, depending on the type of work. In two of the grade school rooms visited, only 0.1 gram of crayon was used in each during the entire afternoon, while in one university class 7.9 gm. were used during a period of 1 hour. It seems reasonable to expect that where a number of students are doing board work, the amount of chalk used would exceed the maximum observed.

To obtain a crayon usage unit for subsequent experiments, the amounts of crayon used by 8 persons writing a standard sentence for a given time were

TABLE I

*Lead Content of Blackboard Crayons*

| *Manufacturer* | *Color* | *Wt. of Stick of Crayon in Grams* | *Per cent Lead* |
|---|---|---|---|
| A * | Yellow | 4.2 | 8.8 |
| B * | Yellow | 10.8 | 7.0 |
| B * | Yellow | 4.9 | 8.9 |
| C | Yellow | 6.0 | 10.3 |
| D | Yellow | 5.7 | 12.0 |
| C | Pale Yellow | 5.8 | 8.4 |
| C * | Pale Yellow | 10.6 | 1.5 |
| C † | Pale Yellow | 10.5 | 0.0 |
| D | Pale Yellow | 5.2 | 5.2 |
| C | Salmon | 5.5 | 6.6 |
| D | Salmon | 4.5 | 1.5 |
| D | (Poster chalk) Salmon | 38.4 | 8.2 |
| D | (Poster chalk) Orange | 34.7 | 4.2 |
| C | Dark Green | 4.6 | 6.7 |
| D | Dark Green | 5.3 | 6.6 |
| C | Light Green | 5.7 | 7.0 |
| D | Light Green | 5.5 | 9.8 |
| D | (Poster chalk) D. Green | 32.1 | 3.7 |
| D | " " Red | 25.0 | 0.0 |
| D | " " Lavender | 30.6 | 0.0 |
| D | " " Brown | 34.7 | 0.0 |
| D | " " Black | 30.6 | 0.0 |
| E | Light Blue | 3.8 | 0.0 |
| A | White | 9.0 | 0.0 |
| A | White | 4.6 | 0.0 |
| E | White | 9.9 | 0.0 |
| F | White | 5.6 | 0.0 |

* Chalks commonly used in place of white in classes for children with deficient eyesight

† A lead-free yellow blackboard crayon recently placed on the market as a substitute for lead pigmented crayons

determined. The average was found to be 0.28 gm. per person per minute of actual writing, with a maximum of 0.58 and a minimum of 0.10 gm. Preliminary experiments also showed that for a given period of writing, the amount of crayon usage from a 5 gm. stick was approximately the same as from one having twice the density.

### DUST EXPERIMENTS

The studies relative to the amount of crayon dust dispersed into the atmosphere were divided into two parts—those made in a room under controlled conditions and those made in actual classrooms.

Air samples were obtained with a Greenburg-Smith all glass impinger using 5 per cent nitric acid solution as the collecting medium. The concentration of lead was determined by the dithizone method of Harrold, Meek, and Holden.[2]

The room used for the controlled experiments was 38 by 14 by 11½ ft. high and was provided with mechanical ventilation. The outlet grill was located to the right of and 6 ft. from the blackboard. Air entered the room through a door at the rear. Atmospheric samples were collected at two points, one adjacent to the blackboard at the breathing zone of the individual writing and the other in the center of the room at a height corresponding to the breathing level of students.

The variable factors in the controlled

tests were crayon used during a given time and the amount of ventilation provided. Crayon usage was varied between 1 and 10 gm. per ½ hr. period. During Series A, the amount of air removed was approximately 1½ cu. ft. per min. per sq. ft. of floor area, and during Series B, ¼ cu. ft. per sq. ft. of floor area.

The data from the controlled experiments are given in Table II. The air samples were collected during two periods, Periods I and II. During Period I, previously weighed amounts of yellow crayon containing 6.8 per cent lead were used in periodic writing for 30 minutes. Air samples were collected adjacent to the blackboard and in the center of the room throughout the period. Immediately following Period I, a second set of air samples was collected for 30 minutes (Period II) at the same locations. No writing or erasing was done during this period. In some cases, a third set of samples was collected beginning 70 minutes after writing had ceased and continuing for 30 minutes.

The data in Table II show that when using 6.8 per cent lead crayons, the concentration of lead in the air may at times exceed the 1½ mg. of lead per 10 cu.m. of air recommended by the U. S. Public Health Service as the maximum amount permissible for prolonged exposure. The data further show that during the writing period, the amount of lead at the breathing zone of the person writing is dependent on the rate of crayon usage. During the writing period the lead was dispersed throughout the room air, but in all cases the concentration was below that at the blackboard. The maximum concentration of lead found in the samples collected 70 minutes after writing ceased was 0.2 mg. per 10 cu.m. of air.

The second group of tests was con-

TABLE II

*Lead Concentration in Air Under Controlled Conditions*

*Concentration of Lead Dust in the Atmosphere Expressed in Milligrams of Lead per 10 Cubic Meters of Air*

| | *Adjacent to Board* | | *Center of Room* | |
|---|---|---|---|---|
| *Grams of 6.8 % Lead Chalk Used During 30 Minute Writing Period* | *Period I Periodic Writing for 30 Minutes* | *Period II 0–30 Minutes After Period I* | *Period I Periodic Writing for 30 Minutes* | *Period II 0–30 Minutes After Period I* |
| | | *Series "A"* | | |
| 1 | 0.5 | 0.3 | 0.3 | 0.2 |
| 3 | 0.9 | 0.2 | ... | ... |
| 5 | 1.0 | 0.2 | 0.4 | 0.2 |
| 10 | 2.2 | 1.5 | ... | ... |
| 10 | 5.3 | 0.3 | 1.9 | 0.4 |
| 10 | 1.3 | 0.2 | 0.6 | 0.2 |
| | | *Series "B"* | | |
| 1 | 0.5 | 0.2 | 0.4 | 0.2 |
| 3 | 0.6 | 0.2 | 0.4 | 0.3 |
| 5 | 0.8 | 0.3 | 0.5 | 0.3 |
| 10 | 1.7 | 0.5 | 0.9 | 0.4 |
| 10 | 2.3 | 1.3 | .. | ... |

TABLE III

*Lead Concentration in Air of Several Classrooms*

| Run Number | Room in Which Test Was Made | Room Conditions Typical of | Mechanical Ventilation | Per cent of Lead in Chalk | Conditions Prior to Sampling Period: Grams of Chalk Used | Conditions Prior to Sampling Period: Period Over Which Chalk Was Used, in Hours | Conditions During Sampling Period: Grams of Chalk Used | Conditions During Sampling Period: Sample Collecting Period in Hours | Concentration of Lead in Atmosphere—mg. of Lead per 10 cu.m. of Air |
|---|---|---|---|---|---|---|---|---|---|
| | | | | *Center of Room* | | | | | |
| 1 | A* | Winter | No | 1.5 | 9.5 | 4 | 0 | 1.4 | 0.2 |
| 2 | B* | Winter | No | 1.5 | 3.0 | 1 | 0 | 1.0 | 0.4 |
| 3 | B | Winter | No | 1.5 | 0 | .. | 3.8 | 1.0 | 0.8 |
| 4 | A* | Spring | No | 6.8 | 8.4 | 4 | 0 | 0.9 | 0.1 |
| 5 | B | Summer | No | 6.8 | 2.9 | 1 | 5.3 | 0.8 | 0.3 |
| 6 | B | Summer | No | 6.8 | 8.2 | 2 | 0 | 0.8 | 0.0 |
| 7 | C | Winter | Yes | 8.0 | 13.0 | 1 | 17.0 | 1.0 | 1.3 |
| | | | | *Adjacent to Board—Writing and Erasing* | | | | | |
| 8 | B | Summer | No | 6.8 | 0 | .. | 2.9 | 0.8 | 0.2 |
| 9 | C | Winter | Yes | 8.0 | 0 | .. | 13.0 | 1.0 | 5.9 |

* Chalks commonly used in place of white in classes for children with deficient eyesight

ducted in 3 classrooms A, B, and C. Due to the noise created by operation of the impinger apparatus, it was not feasible to collect air samples while the classes were in progress. Runs No. 1, 2, and 4 were made immediately after class had been dismissed. The remaining 6 runs were made in classrooms where no classes were in progress.

Classroom A was 74 by 13 by 11½ ft. high, and Classroom B, 20 by 15 by 10¾ ft. high. Neither of these rooms was provided with mechanical ventilation, the only ventilation being that occurring naturally through doors and windows. To vary conditions in room B, the doors and windows were open to different degrees, thereby simulating conditions as existing during winter, spring, and summer. Room C was 29 by 26 by 15 ft. and was provided with mechanical ventilation.

Data from this series of tests are given in Table III. The results were in agreement with those of the controlled group in that ordinarily the concentration of lead in the air at the center of the room was below 1.5 mg. per 10 cu.m. of air. However, under adverse conditions it seems reasonable to expect that higher concentrations of lead may occur. During Run 9, in which 13 gm. of crayon were used in 1 hour's writing, the concentration of lead in the air at the blackboard was 5.9 mg. per 10 cu.m. During Run 7, the concentration of lead at the center of the room was 1.3 mg. of lead per 10 cu.m. of air. Thirteen gm. of 8.0 per cent lead crayon were used prior to Run 7, and 17 gm. during the run. The quantity of crayon appears to be high but on the basis of data obtained from actual crayon usage, indications are that conditions would be comparable to a class of 15 students each writing 7 minutes out of 2 hours.

Individuals subjected to the greatest lead exposure are teachers and students writing at the boards, and janitors cleaning boards, erasers, and classrooms.

When it is considered that children

appear to be more susceptible to lead intoxication than adults, the desirability of replacing lead chromate as a pigment is apparent. In addition to the possibility of lead absorption by inhalation, the danger of ingestion by nibbling pieces of crayon as pointed out by Jephcott should not be ignored. The fact that at least one manufacturer has produced a yellow crayon free of lead shows that the hazard can be eliminated.

## SUMMARY AND CONCLUSIONS

1. Of the 27 samples of blackboard crayons analyzed, lead was found to be present only in various shades of yellow, orange, and green. The maximum quantity of lead found was equivalent to 12 per cent by weight.

2. One sample of yellow blackboard crayon contained no lead.

3. Where colored crayons containing appreciable quantities of lead are used, the concentration of lead in the classroom atmospheres may exceed the toxic limit of 1.5 mg. per 10 cu.m. of air as determined by the U. S. Public Health Service.

4. In view of the potential health hazard involved in the use of lead as a pigment in colored crayons, this material should be replaced by one which is non-toxic.

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## TUBERCULOUS INFECTION DUE TO MILK

WHILE the danger to human beings from bovine tuberculosis is now universally recognized, very seldom have what might be called acute epidemics of tuberculosis been recorded, and still more rarely has the outbreak been traced to a single cow with recent tuberculosis of the udder. Hence the outbreak in Sweden recently reported [1] is of more than usual interest and emphasizes the necessity of vigilance.

In August and September of 1936, in the village and municipality of Horred, Sweden, several children showed tuberculous symptoms for which no human source of infection could be found. By the middle of October, 30 cases had been recorded. Altogether, 56 infected persons were discovered of whom 9 were from 13 to 64 years of age. Among 102 children who did not consume milk from the tuberculous cow there were only 8 positive reactors, while of 32 who were known to have consumed this milk, 29 were positive. Of 29 children under school age, 11 who did not take any of the infected milk all gave a negative reaction, while of 18 who drank the milk, 16 gave a positive reaction. Since 1935 all inhabitants of Horred except those who had their own cows, have been obliged to drink the milk from the Horred dairy. Pasteurized milk was objected to on account of its boiled taste so that the milk for drinking purposes was consumed raw.

A striking point is that the herd was one which since 1935 had been attached to the State subsidized organization for the prevention of tuberculosis in cattle. It had been physically examined in May, 1935, when one cow was taken out on account of tuberculosis. Another examination was made in May, 1936, when no tuberculous animals were discovered. In September, 1936, a third clinical examination was made and at that time samples of milk from the 22 animals of the herd were taken for bacteriological examination. One cow showed clinical mastitis but tuberculosis was not suspected. However, her milk was found to contain many tubercle bacilli demonstrable on centrifugation and staining. This cow was slaughtered at once. Post-mortem examination showed recent tuberculosis, mostly in the right lung and pleura. There were diffuse peritoneal

deposits and a number of mesenteric glands were enlarged and showed caseous foci. The left half of the udder was enlarged and hard, and numerous tuberculous foci were found.

It is plain that milk from a cow with active tuberculosis of the udder was consumed by most of those children who developed tuberculosis, while others who did not drink this milk escaped. There is no statement that tubercle bacilli were isolated from these children and proved to be bovine, though the history justifies this conclusion.

It is clear that clinical examination of cattle to detect tuberculosis is not to be wholly depended upon. This herd was under a State organization whose object was the prevention of tuberculosis in cattle in an infectious form. Presumably more than common care was given to this herd. Apparently the veterinary inspection was done by competent men, yet not until a number of children became ill was it suspected that the herd contained an animal excreting tubercle bacilli in its milk.

The second lesson is that tuberculin tests of cattle supplying milk for market purposes should be made at regular intervals. The third lesson, and perhaps most important, is that all milk for drinking purposes should be pasteurized.

In view of this history it is surprising that the Swedish Parliament in 1938 passed an act regulating the sale of milk and cream for human consumption under which exemption may be granted from compulsory pasteurization of milk not only from cows which do not react to tuberculin, but also from those which have passed a clinical examination. It is clear that under such exemption the users of the milk are not entirely safe, as this tragic outbreak demonstrated. Again the necessity of pasteurization has been shown in a striking manner.

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## A NEW NAME FOR DRY MILK SOLIDS

NEARLY a quarter of a century ago, when skim milk usually was regarded as a waste product, definitions for "skim" or "skimmed" milk and "dried skimmed milk" were adopted by the federal government and most of the states. These official names and definitions usually have remained unchanged to the present day, although the conditions of use and the nutritional appreciation of these products have greatly changed.

In recent times, the so-called dried skimmed milk has become an important article of commerce, and significant as an actual and potential contributor to human nutrition, as well as to animal feeding.

While pasteurized or certified whole milk is properly recognized as our most nearly perfect food, much of its food value is actually below the cream line. Removing the butterfat from whole milk may take away this source of food-energy and the vitamins, A and D, associated with it, but there remain practically all of the valuable minerals, proteins, lactose, and other vitamins of the original milk.

When these important non-fat milk solids are dried, the resulting product is a concentrated and inexpensive source of the food-minerals, calcium and phosphorus, and of milk proteins, milk sugar, and certain vitamins. Dry milk

solids are rich in vitamin $B_2$ (riboflavin) and represent a good source of vitamin $B_1$ (thiamin), as well as of other components in the vitamin B complex.

The most extensive modern use of dry milk solids of this type is in the baking industry, which now purchases about 100,000,000 pounds annually for inclusion in white bread and cake. The growth of this desirable use of milk solids in bread is indicated by the fact that in 1923 less than one-third of this quantity was obtained by bakers. Dry milk solids are also widely employed in the making of confectionery, in certain meat products, and in various other food industries.

Because of the present wide use of dry milk solids and the unfortunate connotations of the words "skim" or "skimmed" in the public mind, it has been suggested that official designations of this product be changed from "dried skimmed milk" to "dry milk solids not over 1½ per cent fat," a definition which already has been adopted in two or three states. Since this terminology would permit of accurate and truthful labelling of a wholesome product, and would tend to remove the popular misconception about the real value of skimmed milk, the proposed new definition seems reasonable and deserves favorable consideration.

## ADVANCE IN FOOD HANDLING

IT is well inside of the truth to say that one of the great problems of the world today is—as it always has been—food. With increasing populations the carry over from the season of plenty—in other words, the preservation of foodstuffs—has grown in importance. The scientific student of nutrition has investigated what happens to foods during the processes necessary. Are their wholesome qualities preserved, destroyed or impaired? The commercial food purveyor has been quick to recognize the scientific aspect. Extensive laboratories are maintained, staffed by outstanding experts provided with every facility for the best work. Volumes are pouring forth on almost every aspect of nutrition but not enough has been told the public about the practical aspects of food preservation. The advances made in handling and preserving foods are fully as notable as the scientific discoveries concerning the foods and equally as necessary for good results. In a very few years we have advanced from the wooden container to stainless steel, which now holds the stage as the best material so far discovered for use in plants devoted to the preparation of foods. It also is largely used in kitchens and pantries on ships as well as on land. It finds wide use for cutting and serving tables and containers for keeping foods hot in eating places for which it is particularly good since foods may be kept for any length of time without damage or change. Breweries are adopting it for storage and for transportation, while piping is largely employed for service in bars.

After many experiments it has been found that a low-carbon steel (0.1 per cent) to which 18 per cent of chromium and 8 per cent of nickel is added is most satisfactory, having a high corrosion-resistance, yet being remarkably ductile and workable. Sheets and plates of such composition can be rolled and bent to shape, are suitable for press work, deep-drawing (milk and ice cream cans, for instance), can be welded by any of the several methods, except hammering, to form smooth and homogeneous joints which can be readily polished. This is important as the material exhibits its best corrosion resisting properties when the surface is free from films or scales. It is amenable to brazing, hard or soft soldering, etc., but these processes are not recommended these days in milk or food plants.

No metals except the noble ones have such a wide range of resistance as stainless steel. It is not attacked by any of the ordinary foods or food acids and as a consequence does not give off to the foods dissolved metal, hence their flavor, color keeping qualities, etc., are not changed. It is very tough and resistant to wear and tear, scratching and distortion, points of importance in the consideration of new installations, since the greatly prolonged life compensates for the higher first cost. The expense of maintaining a clean and hygienic plant is reduced to a minimum. Hot caustic soda does not affect it at all, so can be used for washing without fear of damage. It is the same throughout its mass, not depending on a metallic coating of veneer, consequently there is nothing to peel or wear away.

Certain brines employed in cooling systems, notably calcium chloride brine, are liable, under certain conditions, to pit the steel. This feature has been taken care of by incorporating into the 18 chromium-8 nickel type of stainless steel from 0.5 to 1 per cent of silver, which forms "on the exposed surface of the steel a continuous self-healing film of a silver compound originating from silver which is intra-granularly included in the steel." [1]

So far the milk industry is one of the largest users of stainless steel equipment. In many new plants it is specified throughout. In this country the so-called glass-lined tank is still used for rail transportation over long distances. In England milk is carried to central depots from the farm in "churns" where it is transferred to stainless steel tanks, mixed in other tanks of this material, then pumped into insulated stainless steel rail tank wagons for transportation. In the great city plants the pasteurizers, holders, cooling tanks, filling machines, and bottle platforms are all of stainless steel. In the newest plants for the production of milk powder stainless steel is used throughout.

The increasing use of stainless steel in all the various types of food handling, preservation, preparation, and serving, especially in public places, is to be welcomed as marking a distinct advance from the public health point of view as well as from that of the manufacturer.

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1. *Indust. & Eng. Chem.* (News Ed.), June 10, 1939.
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# BOOKS AND REPORTS

**Medicine in Modern Society**—*By David Riesman. Princeton, N. J.: Princeton University Press,* 1938. 226 pp. Price, $2.50.

Based upon his rich experience as professor of clinical medicine and of medical history, Dr. Riesman here presents a thoughtful interpretation of medicine addressed primarily to intelligent laymen. The purpose of the work is clearly implied in the question posed at the start: "Is it not just as important for the cultured man to know something of the history of the circulation of the blood, of vaccination, of anesthesia . . . as it is to know the story of the Punic Wars . . . ?" It is a sad fact that history, as taught in the schools, still largely omits the vital story of science in general and of medicine in particular, and Dr. Riesman here shows how effectively this theme can be and should be developed.

The book begins with a historical survey which emphasizes the medical progress of the 19th and 20th centuries. The discussion of recent developments in such fields as allergy and nutrition is clear and specific, so that the lay reader will find in it not only cultural background, but also information valuable for his own health. There then follow a number of chapters on special topics, such as cancer, the neuroses, and healing cults. These also are presented in a simple yet authoritative manner. Something of the wisdom of a tolerant medical philosopher creeps into all of these essays. Indeed, it is the blending of scientific with human understanding—of science and art—which affords one of the chief charms of the whole work.

The final sections are addressed to the medical profession as well as to the laity, and contain chapters on medical education, ethics, and the social outlook. In dealing with the difficult and controversial questions involved in "socialized medicine," Dr. Riesman naturally displays a full appreciation of the great services which the medical profession has afforded society, and notes the importance of protecting this group against any changes which would limit its real usefulness. He also faces squarely the lack of medical service available to certain classes, and the consequent need for some type of professional organization to meet this need. Certain of the usual objections made to various plans for "social medicine" he finds untenable; and his final conclusion is that some form of health insurance for the middle classes, under the control of the organized profession, offers the best way out of present difficulties. All in all, this is a most sane and thoughtful book, the sort of work which should be made a part of the training of all well educated citizens.

RICHARD H. SHRYOCK

**The Treatment of Clinical and Laboratory Data. An Introduction to Statistical Ideas for Medical and Dental Workers**—*By Professor Donald Mainland. Edinburgh: Oliver & Boyd, Ltd.,* 1938. 340 pp. Price, $4.50.

The author is a professor of anatomy at Dalhousie University who, in the course of his work, became impressed with the importance of modern statistical methods, and, having searched out such methods for himself, undertakes in this book to give laboratory and clinical workers the benefit of his

findings. The viewpoint and technics advanced in the book follow closely those of R. A. Fisher, thus providing another example of the immense influence that Professor Fisher continues to wield on the newer recruits to the art of statistics. Very largely the book concerns itself with tests for "significance," by which one can "make adequate allowance for chance in all observations." It gently but firmly castigates medical and dental investigators for neglecting consideration of chance variation, and opines that they have therefore frequently gone astray in their conclusions. The reviewer cannot, from his own experience, conscientiously agree that so many casualties are attributable to this particular weakness, but there can be no doubt that good quantitative work should take account of "chance" variability.

In the one illustration of the fallacies which result from the neglect of statistical tests elaborated by the author, we side with the writer criticised. The occasion for criticism is a report on the effectiveness of serum treatment in mitigating paralysis or death during an epidemic of poliomyelitis. The report found a significant difference in the percentage of cases untowardly affected in the treated and untreated groups, but, self-critically, the report said, "A criticism may be raised. Are not the more favorable results observed in the serum-treated cases due to the fact that they are milder cases . . . ? We may answer it from several different angles. . . . Following Draper's view that a cell count in the spinal fluid of over 100 indicates a serious type of disease, a comparison of the cell counts in treated cases and controls shows no appreciable difference in numerical incidence of the counts, from which it would follow that there is no significant difference in the average severity of the two groups of cases." It is this argument in the report that the author criticises. He says, "Now it is possible from the report to test the association between cell count and severity of disease. The table on p. 81 quotes numbers of cases in each instance. In neither set of cases is there a significant association between cell count and severity of the disease; therefore it is not permissible to use the cell count as a criterion of severity in the cases under investigation.

Why is it not permissible? The authors of the report did not in the first place rest their use of the cell count as an index of severity on their own investigation, which had another purpose, but on Draper's, and unless evidence is adduced which casts doubt on the validity of Draper's work, there is nothing relevant here. Do the data obtained by the author from the report and presented as a table (p. 81) refute the conclusion of Draper that there is a correlation between severity of disease and cell count? On the contrary, we find on examining the table that it confirms Draper throughout! In the non-treated group as well as the treated group, the rate of incidence of death or paralysis is considerably greater in the patients with high cell counts. On what then did the criticism of the author rest? Apparently on the fact that the difference between the incidence in the high and low cell count groups was not "significant" in the technical sense that the chi-square P did not reach some arbitrary low level.* But this would be relevant only if the report were trying to prove from its own observations that cell count was correlated with severity of disease. Actually the report rested this con-

* For the experience as a whole, this reviewer finds that the difference in incidence between the high and low cell count groups is +16 per cent, and the probability of such a difference on the assumption of chance variation alone is 0.04. Such a small P would be, for some statisticians, enough evidence to indicate a correlation even if there was no other evidence available!

clusion on Draper's work, not the present investigation, and it does not appear clear to us how the confirmatory findings in the investigation, even though if taken alone they would not be considered sufficient to *establish* the correlation, should be held to cast *doubt* on the correlation previously found.

There are arguments in other parts of the book which similarly overstress the importance of a "P" found from some statistical test, to the exclusion of what appears to us as more important considerations. However, enough has been said to indicate why, in the opinion of this reviewer, though a medical or dental worker will find much that is interesting and useful in this book, it will be hazardous for him to accept it as the last word.

JOSEPH BERKSON

**Alcohol in Moderation and Excess**—*By J. A. Waddell, M.D., and H. B. Haag, M.D. Richmond, Va.: William Byrd Press,* 1938. 184 pp. Price, $1.00.

The General Assembly of Virginia in 1936 adopted a resolution requesting the University of Virginia and the Medical College of Virginia to provide a report on "The effects of the use of alcohol upon the human system in respect to both moderate and excessive use thereof." In consequence, a report was prepared by two professors, one in each of the two institutions named, with the collaboration of six other faculty members. The report was prepared primarily as a source book for school teachers rather than as a text.

When the General Assembly met in 1938 the manuscript was referred to the Senate with the expectation that it would be printed as a Senate document. Prior to the convening of the General Assembly, however, a summary of the report had been made public, and a protest was made by the dry organizations of the state against the printing of the document. It was not printed by the Senate. Later the House ordered that the report be printed, so its members might read it, but the protest of the "drys" was again effective; all copies were ordered burned.

Subsequent to the rejection of the report by the Assembly, the manuscript and copyright privileges were returned to the authors and the book was published commercially. The material in the present edition is the same as that used in the edition burned.

The book first provides material on the properties of alcohol and certain pertinent physiological data. It then deals with the effect of alcohol on the central nervous system, and, somewhat less fully, with its effects on the respiratory, circulatory, and digestive systems.

According to the authors, much of the information on the effect of alcohol is in a state of flux and so, unfortunately, many vital questions still remain debatable. The authors have endeavored to cull reasonably conservative deductions from the studies at hand, and the volume seems to deserve the opinion of the Virginia State Board of Education which describes it as a most valuable contribution, scientifically sound and very scholarly.

REGINALD M. ATWATER.

**Educating for Health: A Study of Programs for Adults**—*By Frank Ernest Hill. New York: American Association for Adult Education,* 1939. 224 pp. Price, $1.25.

One of a series of studies in the social significance of adult education in the United States, this book describes the work of important organizations engaged in teaching American men and women to lead healthier lives. "Health education is carried on by such a multitude of public and private agencies," states the foreword, "and is so varied

in character, that it forbids a thorough treatment in a single volume."

This volume aims to give an over-all view of what is being done, indicating interrelationships, trends, and problems. The material was gathered from visits to a dozen states, many cities and counties, by a careful, critical but apparently sympathetic observer, who was primarily concerned with health education not as an aspect of health work but as a form of teaching and learning. The author has done a constructive job and rendered a valuable service from which public health educators and administrators may derive benefit. IRA V. HISCOCK

**Health for 7,500,000 People**—*Annual Report of the Department of Health, City of New York, for 1937, and a Review of Developments from 1934 to 1938. New York: Department of Health,* 1939. 390 pp.

This volume is more than an annual report; it is a well written review of the administrative and physical development of the Department of Health of our largest city during the 4 year period 1934 to 1938. Attractively bound and printed, with numerous charts and photographs, its informal style and simplicity of presentation insure its readability, not only to technical workers in the field, but to the public at large.

Departmental activities are outlined in four sections, the administrative section, the medical, nursing and bacteriological section, the sanitary section and the vital statistics section. Under those wide headings separate services are discussed in short chapters. Perhaps the most interesting of these is that dealing with development of the neighborhood health work. Although plans had been worked out previous to the beginning of the period under review, only one of the 30 districts into which the city had been divided had been supplied with a health officer. In 1934 the positive initiation of the program began with the appointment of administrative staffs for seven districts. The beginning of 1938 found 13 civil service district health officers appointed as well as 7 medical officers-in-charge assigned to district work. Nine new health centers had been built and a tenth building remodeled for this purpose.

Other chapters describe the anti-syphilis campaign, reorganized tuberculosis work, pneumonia program, personnel training program, modernization of the bureaus responsible for supervision of food supplies and environmental hygiene, and the work carried on in the new William Hallock Park Laboratory. One chapter is given to the place and use of federal grants in the health program.

The vital statistics section of the report adequately presents the main features of natality, infant and maternal mortality, and mortality in general. Standardized death rates are available by cause as well as rates by sex, color and age. In addition a number of repository tables have been included which give detailed data on trends of the principal causes of death and the sex and age distribution of deaths by cause. J. H. WATKINS

**Vitamins and Vitamin Deficiencies**—*By Leslie J. Harris, Ph.D., Sc.D. Philadelphia: Blakiston's,* 1938. 204 pp. Price, $2.50.

This is the first volume of a series to be published under the general title of Vitamins and Vitamin Deficiencies. This volume is entitled Vitamin $B_1$ and Beri-beri—Introductory and Historical.

This is a very interesting little book. The author states that his aim has been to provide a "comprehensive synopsis." He has done this.

An enormous amount of research is reviewed but there is no attempt to

give detailed information on any point; an excellent bibliography accompanies each chapter. There are a good many illustrations which add greatly to the value of the book. Pictures of 8 outstanding pioneers in the study of vitamins are given: Lind, Takaki, Eijkman, Grijns, Holst, Hopkins, Funk, and McCollum.

The following sentence taken from the Foreword by Sjr F. Gowland Hopkins is a fitting close to this brief review:

> I can strongly recommend the book to all students who desire acquaintance with the present position and outlook in the field of vitamins, and believe that the specialist will welcome an admirable summary of the facts available to-day and a bibliography fully up to date.

NINA SIMMONDS

**Macmillan's Modern Dictionary**—*Edited by Bruce Overton. New York: Macmillan,* 1938. 1466 pp. Price, $3.00, with thumb index, $3.50.

The "Modern Dictionary" must be an efficient guide to the choice and in the use of words; it must be concise and compact, yet comprehensive in compass; it must excite the interest of the user and intrigue him to the study of words as living things; and it must by and of itself help in the development of the dictionary habit."

This excellent objective has been attained to an unusual degree. The Editor has given us something really new in dictionaries.

At first one misses the appendixes, tables of various sorts, lists of foreign words, pronouncing gazetteer, abbreviations and signs used in writing and printing, thermometric scales, etc., so often found. Macmillan's uses "one vocabulary that is peculiar to this dictionary," and in alphabetical order one finds a wealth of biographical sketches, geographical names, foreign words and phrases, and indeed information on practically every subject that one can desire. An unusual number of idiomatic phrases are given. Slang is treated kindly in recognition of the growing character of our language, but is properly labelled as such. Many colloquialisms are given.

A decided departure is the omission of illustrations. The editor believes that they have been used to embellish the pages rather than to clarify definitions, and points out that great areas of knowledge such as mathematics, philosophy, psychology, theology, sociology, and other department of human intelligence, although contributing a much larger vocabulary, are scantily illustrated.

The only practical way to judge a dictionary is to use it day by day. By this test the reviewer has found it modern in every respect, easy to use, well printed and helpful. A curious omission is the word "pyrology," the derivation of which seems plain, but for which there seems to be little agreement as to definition. The *Scientific Monthly* for June, 1939, uses the word to mean the "science or technology of fire control."

Macmillan's can be recommended to those for whom it is designed—businessman, speaker, writer, general reader or student. MAZŸCK P. RAVENEL

**Getting Ready To Be a Father**—*By Hazel Corbin. New York: Macmillan,* 1939. 48 pp. Price, $1.25.

This little volume is so interestingly written, effectively illustrated and printed, that one is likely to read it through at one sitting and wish that every man who is "expecting a baby" might have a copy to profit by the experience of George Halladay, the hero of this story. Here is contained useful information given in classes for prospective fathers dealing with maternity care from the father's point of view, selection of hospitals, physicians and nursing care, guides in meeting the physical

and psychological problems to be faced by both parents, expenses, and suggestions for fitting the baby into the home, even to making furniture and equipment which will facilitate the care of the baby in congested quarters.

IRA V. HISCOCK

**Water Supply Engineering**—*By Harold E. Babbitt and James J. Doland. (3rd ed.) New York: McGraw-Hill,* 1939. 690 pp. Price, $6.00.

In any rapidly advancing field of effort having the scope of water supply engineering, it is difficult to keep a textbook current and still not exceed a useful and convenient size. This third edition by Babbitt and Doland seems to have surmounted this difficulty.

Containing 635 pages of text and averaging almost 1 figure to every 2 pages, it covers water works finances, hydraulics; rainfall runoff and consumption; ground and surface waters with methods and devices for developing and purifying them for consumer use; the relations between water quality and diseases; and an interpretation of numerous analytical procedures available today.

As a textbook, it must necessarily be complete. This forces brief treatment of subject matter but notwithstanding that, this new volume fulfils its purpose adequately and well.

ARTHUR P. MILLER

**Population, Race, and Eugenics**—*By Morris Siegel, M.D. Hamilton, Ont., Can.: The author, 546 Barton St., East,* 1939. 206 pp. Price, $3.00.

This is a good discussion of the problems of race and eugenics for the lay reader. It treats these problems simply and clearly. It is not overdogmatic nor is it as biased as is often the case when the occasional writer tackles general questions of this kind. It can be heartily recommended to the general reader who would like to get a bird's-eye view of the eugenic situation.

The word "Population" in the title gives the impression that it treats of population questions, aside from "Race" and "Eugenics," more than it actually does, but this does not constitute a serious defect considering the purpose of the book and the public it is intended to reach.

A little more careful proof-reading would have helped it in places.

WARREN S. THOMPSON

**A Textbook of Applied Microbiology and Pathology**—*By Thurman Rice, A.M., M.D. (2nd ed.) New York: Macmillan,* 1939. 271 pp. Price, $2.50.

The only important change made in the second edition of this very practical and usable text for student nurses is the inclusion of a section on General Pathology.

The material on Bacteriology has not been shortened—in fact, it appears in almost the exact form given in the first edition. By condensing the size of the type and omitting a few illustrations the actual number of pages remains the same even though approximately one-sixth of the second edition is given over to the section on Pathology, which covers, after an introduction, Congenital Abnormalities, Neoplasms, Progressive and Retrogressive Changes in Tissues, Inflammation, Circulatory System, and Circulatory Disturbance.

This additional material is presented in the same simple and interesting style which characterizes the older sections. It is not complete enough to serve as a text in a course in General Pathology, but it does supply fundamental knowledge of disease and fulfil the purpose for which it was intended. The usefulness of this added section will largely depend upon the organization of the course for which the book is used.

The drawings have been made by the

author. In most cases they are exaggerated as to size in order to emphasize special features.

The book will continue to hold its high place among texts of this type.

ANNA DEAN DULANEY

**Class Fertility Trends in England and Wales, 1876–1934**—*By John W. Innes. Princeton, N. J.: Princeton University Press*, 1938. 145 pp. Price, $2.50.

That there exist wide differences in fertility between different social and economic classes in the population has been noted by nearly all population students since the time of Malthus. It has also often been noted that the decline of birth rates during the past half century or more seems to have widened these differences. The matter of larger families among the poor than among the more fortunate social classes has long been of concern to both eugenists and to some sociologists. Recently, however, there have been accumulating scattered bits of evidence which suggest the hypothesis that such fertility differences may be temporary phenomena and that the future may see a closing of the gap between the birth rates of different occupational and social classes. Some students of population are even predicting the complete disappearance of fertility differentials within the next few decades. It was to test this hypothesis of "democratization" of birth rates that the present research was undertaken.

The first part of Dr. Innes's study consists of a careful re-analysis of fertility rates by occupational classes for England and Wales. The data are drawn from the Census Report of 1911 on the Fertility of Marriage in England and Wales by the late T. H. C. Stevenson. The results of this analysis seem to point rather clearly to the conclusion that the sharp decline in fertility which seems to have begun about 1876, proceeded at quite different rates for different occupational groups. This divergence in fertility trends for different social classes continued unabated up to the turn of the century, so that by 1900 fertility rates were both absolutely and relatively further apart than they were 25 years before that time.

In the second part of his study, the author attempts to pursue the analysis of class incidence of fertility decline beyond the period for which the report of 1911 supplies the data. Since later census reports do not provide material which is comparable with that found in the earlier report he was forced to have recourse to another approach and method. To study class differentials in fertility beyond 1911, use is made of fertility data for different boroughs and districts of metropolitan London classified into "class-areas" on the basis of socio-economic criteria. If we may accept London area-class fertility rates as comparable with English occupational class fertility rates, then the pattern of class differentials has been quite different since 1900 as compared with the earlier period. Already by 1911 the lead in the decline of the birth rate had passed from those classes of highest socio-economic status to those lower in the scale. By 1934 all fertility differences between the three higher classes had been obliterated, and the differential between classes I and IV had been reduced to an insignificant amount. Class V, that with the lowest social and economic rating, continued to retain the position of highest fertility, but the absolute difference between it and the other classes had greatly diminished and its downward fertility trend relatively had kept pace with the trends of other classes. Between 1931 and 1934 the sharpest decline in fertility was found in class V.

It would indeed be foolhardy to con-

clude from the results of this and a few other similar studies that differences in class fertility will disappear within the near future. The author warns against drawing any such conclusions even for London, to say nothing of all England and Wales. It may not be too much to say, however, that the study does give us reason to discount the alarms of the eugenists in their concern over the future quality of the population as a result of differential birth rates. Certainly the evidence presented in this study does not seem to indicate that lack of "prudence and foresight" is reproducing a special "social problem" class in the population.

Dr. Innes has made an important contribution to the literature on differential fertility. While the lack of comparable data may preclude the possibility of repetition of his methods in other countries, his study should stimulate researches elsewhere for the testing of the hypothesis of the "democratization" of the birth rate.

C. T. PIHLBLAD

**The Health of College Students**—*By Harold S. Diehl, M.D., and Charles E. Shepard, M.D. Washington: American Council on Education,* 1939. 160 pp. Price, $1.50.

This book is a report by the authors to the American Youth Commission of the American Council on Education. It is a study of 4,800 young people and 500 institutions of higher learning. Through the CCC the Council obtained data on a large number of young men from the lower economic backgrounds. The result is a most interesting book, giving what we believe to be the most comprehensive study yet made of the population of this class and age.

It opens with a general consideration of the health of youth, goes on to give the development of college health work, discusses the problems, programs, and organization of such services, and ends with Suggestions for Organization and Development of College Student Health Service. Three appendices give statistical tables, forms used in study, and a list of the colleges and universities which participated.

The work has been carefuly done under competent direction and contains a wealth of material of great value. It is open to the criticism that a great deal of the material was obtained through questionnaires, and allowance must be made for the human disposition to present a good appearance.

The report can be thoroughly recommended to all interested in this great question and should find a place in every university and college library. The printing and binding are excellent.

MAZŸCK P. RAVENEL

**Nursing Through the Years**—*By Corrine Johnson Kern. New York: Dutton,* 1939. 340 pp. Price, $2.50.

The story has its beginning in the operating room of a well known San Francisco hospital as Corinne Johnson, the author, is on duty for the last time as a student nurse. The next day she is to start private practice, she has thoroughly enjoyed her student days and wonders if she will be successful and happy in her chosen life's work.

In the spring of 1900, she was employed as office assistant to Martha Purdy, M.D., who was lovable, queer yet the most tactfully impertinent human being imaginable. The doctor's life was filled with purposeful undertakings—births, surgery and medical care—the days were not long enough to care for her busy practice in that remote country town.

Her next assignment was in a country home near the Pacific Ocean. At first the attending physician was disappointed to find that a young nurse weighing only ninety pounds was sent in answer to his request, but he was more than delighted with her capable

handling of a family situation which resulted in his patient's rapid recovery.

Interwoven with the personal account of caring for patients in all walks of life from the homes of the uneducated and poor to homes of refinement and of the very wealthy, is the story of the progress in the fields of medicine and nursing.

Corrine Johnson won the respect of all with whom she associated; her happy, pleasing personality brought encouragement and hope as well as qualified nursing service to each one.

She retired to her mountain bungalow to rest but found that her chosen career of nursing suffering humanity was to keep her busy, so she continued to answer their calls any hour of the day or night.

The story is simply and sincerely told and will interest lay readers as well as professional members.

MARGARET STRONG

**Man and His Health**—*New York: Exposition Publications, Inc.*, 1939. 96 pp. Price, $.50.

The American Public Health Association has a special interest in the Medical and Public Health Exhibits at the New York World's Fair. In the late 1920's planning for an exhibit of this sort began. In 1931 a committee was appointed, and when the World's Fair was announced in 1935 this committee saw its opportunity and succeeded in interesting the Fair Corporation so that one of the most interesting exhibits at the World's Fair is in the Medicine and Public Health Building which contains the Hall of Medical Science and the Hall of Man. The exhibits are the result of the combined efforts of nearly 1,000 individuals.

The Hall of Man was designed to house the original Oberlaender Trust Collection of exhibits on anatomy and physiology. It was developed and sponsored by the American Museum of Health. The exhibit is to satisfy Man's natural curiosity about himself, to introduce Man to Man himself. Here the anatomy and physiology of the body is illustrated, showing how man breathes, walks, digests his food, how his senses operate, and how he reproduces himself. It contains a Transparent Man of life size, showing the relation of the skeleton and organs of the body. A remarkable series of models has been developed for this purpose.

In the Hall of Medical Science are many exhibits of interest to everyone, only some of which can be mentioned, but enough to show the range of subjects of wide general interest which they cover: Maternal Health, Child Health, Normal Growth and Development, Veterinary Medicine and Public Health, Medical Education, Housing for Health, Syphilis, Infections, Immunity, and Heart and Circulation of the Blood. These have been sponsored by voluntary associations, such as the American Medical Association, American Public Health Association, American Social Hygiene Association, American Veterinary Medical Association, etc., as well as by some foundations, and a certain number of commercial firms interested in public health and the manufacture of medicinal preparations.

Each one of these exhibits is under an Exhibit Committee made up largely of persons interested in the special lines to which the exhibit refers.

The American Museum of Health, incorporated as a nonprofit educational institution under the laws of the State of New York, was created in December, 1937, and makes its début at the New York World's Fair. Even before its dedication, June 17, more than 1,750,000 had examined the displays which it contains. The interest shown has exceeded all expectations. The day-to-day attendance runs more than a third of the paid admissions to the Fair.

The American Museum of Health has created and acquired most of the important and durable exhibits now on view in the Medicine and Public Health Building. At the conclusion of the Fair it is planned to move these exhibits to a permanent institution for visual health teaching in the City of New York. Coöperation in securing a building for the purpose has been assured.

MAZŸCK P. RAVENEL

**The Comparative Values of Chest Roentgenograms Made on Film and on Paper for Industrial Surveys**—*Medical Series, Bulletin No. II. Pittsburgh: Air Hygiene Foundation of America, Inc.*, 1939. 20 pp., 7 charts in pocket.

This study was directed by Dr. E. P. Pendergrass of the Hospital Department of Radiology, University of Pennsylvania, while the technical aspects were developed by the staff of the Moore School X-ray Laboratory. Several leading X-ray companies and film manufacturers coöperated in the project. Of special interest, 49 roentgenologists in eastern and middle western states examined the roentgenograms and recorded their opinions in special questionnaires.

The *Bulletin* discusses The Physical Characteristics of Roentgenograms on X-ray Film and X-ray Paper, Preliminary Experimental Procedure, Roentgenographic Exposure Techniques, Test Roentgenograms and Questionnaires, Physical Data of Test Roentgenograms, Roentgenologists' Comments on Test Roentgenograms, and Conclusions.

While, as has been found before, the average density and contrast of roentgenograms made on paper are inferior to those on film and the former should not be used in place of the latter unless for important practical reasons, nevertheless, if a large group of employees are to be examined by single roentgenograms, the use of paper in rolls is less expensive, quicker, and available for survey work, if the technical methods here outlined are used, with provision for pathological and doubtful cases to be referred for more thorough radiological examination.

Thirty-one of the 49 roentgenologists were of the opinion that the paper roentgenograms would be satisfactory for a preliminary survey. Certain improvements in the technical procedures with paper roentgenograms can be made. The authors believe that since the paper roentgenograms are in no way superior to those on film, it would be best in the long run to attempt to work out a scheme for making roentgenograms on film quickly and at low cost, possibly by using X-ray film in rolls. Decreasing the size of the roentgenograms would also reduce the cost.

In particular, it would be extremely useful to devise a detailed specification of all the technical factors involved in making chest roentgenograms which might be accurately followed thereafter in any laboratory.

The *Bulletin* is necessarily quite technical, and in some places not very clear in language, while considerable basic knowledge of roentgenography and photography is assumed.

EMERY R. HAYHURST

**Education of the Handicapped**—*By Merle E. Frampton and Hugh Grant Rowell. Vol. I, History. Yonkers-on-Hudson, N. Y.: World Book Company*, 1938. 200 pp. Price, $2.40.

The chief virtue of this volume seems to be that it has gathered between its two covers a mass of miscellaneous information concerning the history of care for those handicapped in vision, hearing, speech, mental or social development, and for the crippled and

those "under vitalized" through tuberculosis, heart disease, or malnutrition. Such a compilation fulfils a long felt need and this volume will be welcomed by teachers and lecturers in several fields.

Certain details of the attitudes toward each of the handicapped groups, of the development of specific educational methods for each group, and of the leaders in those movements which have helped or hindered each are recorded.

The authors emphasize how much the handicapped have depended upon the development of social and humanitarian movements and how any progress in their education has *followed* rather than *led* the cultured and scientific patterns through the ages. They conclude that because of the complex nature of needs of these specialized groups this must continue to be so.

A second premise is that the history of these groups has been influenced more by great personages than by social, economic, religious, or political events and, therefore, with the exception of the socially handicapped, the general pattern of telling of the lives and influence of various figures prominent in the history of each group has been followed. This results at times in a succession of names and events in which the theme of the story seems to be only faintly heard. Would it not have been possible to tell as integrated a story for the visually handicapped, for example as Sanford Bates has written on the socially handicapped.

The volume has been edited and apparently largely written by Drs. Framton and Rowell. Distinguished contributors are listed, though credit is given only to Bates in the chapter on the socially handicapped and to Romaine Prior for the chapter on the orthopedically crippled. A bibliography of important books and magazine articles on each subject is included—but the text itself is not documented.

For the public health worker, the volume presents a broad point of view on the subject of the handicapped: one emphasizing the need for and possibility of assisting these persons to live effectively in the world as they find it. To include the cardiacs as well as the blind in the picture is helpful. The story of the former is limited—but the discussion of open air classes is certainly pertinent. No mention is made of other medically handicapped, the asthmatics, for example.

LEONA BAUMGARTNER

**Fighting for Life**—*By S. Josephine Baker, M.D. New York: Macmillan*, 1939. 246 pp. Price, $2.75.

This is the autobiography of one who has gloried in fighting for good causes. Dr. Baker tells of a day when she was 6 years old and all dressed up for some great occasion. She wore a "beautiful white lacy dress with a blue sash and light blue stockings and light blue shoes." As she sat on a horse block contemplating her fine appearance, a little black girl happened to come along, looking peaked and hungry and attired in rags. Thereupon Josephine took off all her own clothing, even her underclothes, and gave them to the poor colored child, returning to her home quite naked. This action Dr. Baker describes as characteristic of herself through life. She says she has always been trying to do things about hopeless situations.

Happily, most of Dr. Baker's struggles ended in victories. Her experience as a pioneer in obtaining medical training for women, and as a militant leader in the suffragist movement, and, above all, her notable career in the New York City Department of Health where she became head of the Bureau of Child Hygiene, are told with detail and zest. The names of many prominent members of the

medical profession, the stage and political life, and scenes and incidents relating to New York, particularly in the nineties and early part of the present century, adorn the pages. The keen enjoyment which seems to have been taken in writing the book is certain to be shared by readers.

It is unfortunate that this entertaining book is marred by a number of inaccuracies. Two examples will suffice.

Dr. Baker's early home was in Poughkeepsie, where she lived until she was 16 years of age. She left, following the death of her father from typhoid fever. The prevalence of the disease that winter, she says, led to the installation of the first municipal water filtration plant in the United States. The truth is that the Poughkeepsie filter was built in 1872 from designs by James P. Kirkwood. If Dr. Baker was 16 then, she must be 83 now, an imputation which she would probably resent.

The discovery of Typhoid Mary is described, but quite incorrectly. Dr. Baker was at the time an inspector in the New York City Health Department and succeeded in arresting the woman with the help of four policemen, after she had been located and her pugnacious character explained. Contrary to Dr. Baker's account the investigation which led to the arrest was not made in order to test any statement of Robert Koch's, nor was it made by anybody connected with the New York City Department of Health, nor any other health department, for that matter. Nor was the arrest made to see whether the investigator's * suspicions were justified. The case had been fully proved by him before it was brought to the attention of the city health department. The effect of Dr. Baker's statement is not only to rob the investigator of much of the credit which properly belongs to him, but gives what seems to be authority to an erroneous account of what has long been regarded as a most important epidemiological discovery. GEORGE A. SOPER

* George A. Soper (Editor).

**Pulmonary Tuberculosis in Adults and Children**—*By James Alexander Miller, M.D., and Arvid Wallgren, M.D. New York: Nelson,* 1939. 193 pp. Price, $3.50.

The authors have republished in book form, with some modification, their articles in the *Nelson Loose-Leaf Medicine.* Both authors recognize the extreme complexity of tuberculosis and the multiplicity of factors which may determine the course of the disease. Miller classifies pulmonary tuberculosis as primary, prephthisical and phthisical. Wallgren as primary, secondary and tertiary, thus following German and French terminology rather than that commonly used in the United States. They feel that these groupings help clarify the pathogenesis of the disease and bring into proper relief the hematogenous spread of the bacilli in the body. Both are critical of the American emphasis on exogenous reinfection, since they feel it to be of minor importance, hence they do not approve of the classification of Primary Tuberculosis and Reinfection Tuberculosis adopted in the 1938 Edition of *Diagnostic Standards,* published by the National Tuberculosis Association. It is the reviewer's understanding, however, that this classification, as explained in *Diagnostic Standards,* has met with very little adverse criticism in this country. Until the reasons for the protean nature of tuberculosis are known with more certainty it is doubtful whether further refinements in classification will have value clinically.

It is natural that the authors, holding the views indicated, do not stress the protection of infected persons from contact with open cases. Indeed,

Wallgren goes so far as to say "it is generally found that children who are tuberculin-positive do not run any risk if they are exposed to tuberculous infection." This is, of course, opposed to the view of tuberculosis specialists and public health workers in this country.

Wallgren discusses in an interesting way erythema nodosum and serofibrinous pleurisy, both of which appear to be more frequent in Sweden than in the United States, and writes out of wide experience of the prognostic aid of repeated sedimentation tests. He states that the demonstration of tubercle bacilli in the gastric lavage water "has no prognostic significance nor does it offer any guidance in treatment or in judging the contagiousness of the child."

Some of the reproductions of X-ray films do not show clearly the pathology indicated in the text. Few typographical errors were noted. On page 37 and again on page 58 the sentence following (3) beginning "the first intercostal spaces between the clavicles" presumably should read "below" instead of "between." At the top of page 153 column 1 "a manner of chance" should read "a matter of chance." Wallgren's rather extensive references are mostly to continental European writers.

For an interesting presentation of the pathogenesis of tuberculosis the book is to be recommended. For details of treatment, especially collapse therapy, other authors dealing comprehensively with therapy should be consulted.

JOHN H. KORNS

**Nutrition**—*By Margaret S. Chaney, Ph.D., and Margaret Ahlborn, M.S. (rev. ed.) Boston: Houghton Mifflin,* 1939. 436 pp. Price, $3.00.

This text has been substantially rewritten since the first edition, published 5 years ago, but the original form has been retained.

*Nutrition* deals with the health of the individual as related to food and the ability of the body to utilize food. Pre-parental education, as that given, should aid considerably in correcting bad food habits and in the promotion of an optimal state of health in the members of the family. Some mention is made of the relationship of food to certain pathological conditions in which diet is of primary importance.

There are 14 excellent chapters discussing energy metabolism, minerals, vitamins, body regulators, nutrition during the reproductive period, and optimal nutrition for infants, children, and adults. Recent developments are presented and an up-to-date bibliography is appended to each chapter. The text is supplemented by 56 fine illustrations and 85 tables. The appendix contains many excellent food value tables, height-weight charts, and other similar data. Its unique value lies in its presentation of an outline of methods for animal experimentation applicable to vitamin testing. This section includes methods for feeding and breeding stock animals; assaying for vitamins A, $B_1$, C, D, and riboflavin; anemia testing; and methods for the preparation of constituents of experimental diets. Detailed directions are also given for the use of the Benedict-Roth recording metabolism apparatus.

*Nutrition* is a well organized book whose authors have overcome some of the failings of similar texts. It deserves a place in the reference library of doctors, nurses, dieticians, and other nutritionists.

C. R. FELLERS AND A. S. LEVINE

# A SELECTED PUBLIC HEALTH BIBLIOGRAPHY WITH ANNOTATIONS

RAYMOND S. PATTERSON, PH.D.

**Telling the Public**—So many of your well read lay contacts will have discovered this excellent dissertation on chemotherapy with sulfanilamide and its derivatives, that you had better prepare yourself to discuss the matter by reading the article, too. Once more Fortune does an outstanding job in public health education.

ANON. Cure by Chemicals. Fortune. 20, 3:42 (Sept.), 1939.

**Health Education by Doing**—In Syracuse (New York) the juniors and seniors in public and parochial high schools are offered the tuberculin test and X-ray examination if positive: about half accept the test, but all learn about tuberculosis. Four years ago a third were positive reactors; today less than a quarter. Discussants suggest that where the tuberculosis rate is low and, as in Syracuse, where community case finding is adequate, justification for school testing may depend upon cost per diagnosis.

AYLING, W. E. The Value of Tuberculin Skin Tests in a School Program. New York State J. Med. 39, 15:1463 (Aug. 1), 1939.

**Historical Note**—Some interesting historical highlights on the evolution from law enforcement to health guidance are told in a brief radio talk by one who took an active part during the whole period. Not many of us can quote what he heard Koch say to Biggs.

BOLDUAN, C. F. Education versus Compulsion. New York State J. Med. 39, 15:1505 (Aug. 1), 1939.

**All about Colds and Related Ills**—Here, you will find a good workmanlike review, brief and to the point, of everything you will need to use in telling your public about the respiratory infections and related conditions. It is a continued story and both parts should be read and saved.

CECIL, R. L. The Control of Respiratory Infections. Preventive Medicine (New York Academy of Medicine). 9, 3:50 (June), 1939.

**Quaranto Die**—Discrepancies between established facts about communicability of certain diseases and the control measures adopted by the Canadian provinces (which in many instances are not as incongruous as our own) appear to the public as evidence of blundering and inconsistency. Health departments may well experiment but there should be better international and interstate agreement in practices.

EMERSON, H. Current Practice in Communicable Disease Control. (and) JENKINS, R. B. The Value of Present Control Measures in Communicable Diseases. Canad. Pub. Health J. 30, 7:325 (July), 1939.

**Putting the Bacterium in His Place**—Useful definitions of some badly abused words — disinfection, sterilization, bacteriostasis — introduce a discussion of practical disinfection methods available for the public health nurse working under the handicaps of the usual home. It's to be continued.

FROBISHER, M. Disinfection in the Home. Am. J. Nurs. 39, 8:833 (Aug.), 1939.

**Some Slips Twixt Cup and Lip**—The time has come, the author says, for health and water works officials to give more consideration to what happens to the water in public systems en route to the consumer in order to minimize health hazards that now exist. What these hazards are and

what to do about them are the subjects of the useful paper.

GORMAN, A. E. Potential Health Hazards in Distribution of Water. J. Am. Water Works A. 31, 7:1143 (July), 1939.

**Limitless Possibilities of Isotypes**—Some information about Neurath's methods that all health educators, in name or in fact, will want to store away for future use.

KAEMPFFERT, W. Facts March On—with Neurath. Survey Graphic. 28, 9:538 (Sept.), 1939.

**Just as One Would Assume**—Nutritionists will appreciate this exact study, the findings of which are that bone development in the wrist is related to the diet of the child. Youngsters on an institutional diet supplemented with reconstituted evaporated milk or with cod liver oil made better bone growth than those who received no supplements.

MACNAIR, V. Effect of a Dietary Supplement on Ossification of the Bones of the Wrist in Institutional Children. Am. J. Dis. Child. 58, 2:295 (Aug.), 1939.

**Cancer Statistics**—This is a significant finding: cancer of the digestive tract which causes about half the male deaths and a third of the female deaths from cancer, makes up only a quarter of all lesions in living cases. In the case of cancer of the breast and uterus the rates are about the same in living and fatal cases. Cancer of the skin and lip are the most frequent sites among males, whereas cancer of the breast and genital tract are most common among females.

MOUNTIN, J. W., *et al.* The Incidence of Cancer in Atlanta, Ga., and Surrounding Counties. Pub. Health Rep. 54, 28:1255 (July 14), 1939.

**Has Today's Tuberculous Patient a Better Chance?**—Some pretty gloomy statistics about the success of tuberculosis treatment have been published recently and annotated in these pages. Now we can report something on the brighter side. The fatality rate has been steadily reduced at Trudeau Sanatorium, and early diagnosis by means of the roentgenogram has played an important part in the decline.

SAMPSON, H. L. Fatality Rates in Pulmonary Tuberculosis. Am. Rev. Tuberc. 40, 1:71 (July), 1939.

**Let Us Flee Said the Fly, etc.**—Here is something you probably do not know: most insecticides used for spraying rooms are toxic and irritating to the skin in sufficient concentration. Both the petroleum distillate and the active principle usually are irritating but no notice to this effect is given. Users should be warned and the directions should urge the use of protective clothing, the exposure of the skin as little as possible, and that containers be kept away from children.

SCHWARTZ, L., and WARREN, L. H. Dermatitis Caused by a New Insecticide. Pub. Health Rep. 54, 31:1426 (Aug. 4), 1939.

**Overweight and High Blood Pressure**—In a large group of physical examinees, especially among those from 50 to 59, overweight was found to increase the incidence of hypertension. The incidence, however, was not as great as some other observers have reported.

SHORT, J. J., and JOHNSON, H. J. An Evaluation of the Influence of Overweight on Blood Pressures of Healthy Men. Am. J. M. Sc. 198, 2:220 (Aug.), 1939.

**Health Officers and Medical Care**—If the public provision of adequate medical care is to come, as this author believes it will, then the established health departments should administer it. If the decade following 1940 is to be a period of accomplishment as the 30's were of discussion, then public health had better set its house in order.

SINAI, N. The Present Situation Regarding the Adequacy of Medical Care in the

United States. Canad. Pub. Health J. 30, 7:319 (July), 1939.

**Among Our Blessings**—At least one worry that is not likely soon to plague American sanitarians is given a thorough airing for the benefit of our British brethren. The title tells the story sufficiently for our purposes.

WILLIAMSON, A. B. The Problem of the Disposal of the Dead after Air Raids. Pub. Health. 52, 11:329 (Aug.), 1939.

## BOOKS RECEIVED

TEXT-BOOK OF MEAT HYGIENE. By Richard Edelmann. 7th ed. rev. by John R. Mohler and Adolph Eichhorn. Philadelphia: Lea & Febiger, 1939. 463 pp. Price, $5.50.

TEXTBOOK OF MICROBIOLOGY. By Kenneth L. Burdon. 2d ed. of A TEXTBOOK OF BACTERIOLOGY. New York: Macmillan, 1939. 638 pp. Price, $2.75.

ELEMENTS OF STATISTICAL REASONING. By Alan E. Treloar. New York: Wiley, 1939. 261 pp. Price, $3.25.

BRUCELLOSIS IN MAN AND ANIMALS. By I. Forest Huddleson. New York: Commonwealth, 1939. 339 pp. Price, $3.50.

HANDBOOK OF CHEMISTRY. By Norbert Adolph Lange. 3d ed. rev. Sandusky: Handbook Publisher, 1939. 1543 pp. Price, $6.00.

BACTERIAL METABOLISM. By Marjory Stephenson. New edition. New York: Longmans, 1939. 391 pp. Price, $7.50.

THE STORY OF SURGERY. By Harvey Graham. New York: Doubleday, 1939. 425 pp. Price, $3.75.

THE BUILDING OF A NATION'S HEALTH. By Sir George Newman. New York: Macmillan, 1939. 479 pp. Price, $6.00.

CHILD TRAINING AND PARENT EDUCATION. References to Material in Recent Books. By Lucile Reiner Stebbing and Caroline Shurtleff Hughes. 2d ed. rev. New York: Wilson, 1939. 83 pp. Price, $.90.

PUBLIC HEALTH IN SOUTH AFRICA. By E. H. Cluver. 2d ed. Johannesburg: Central News Agency. 1939. 342 pp. Price, $4.00.

PUBLIC WATER SUPPLIES IN VIRGINIA. Descriptions and Chemical Analyses. Richmond: State Department of Health, 1939. 159 pp.

# ASSOCIATION NEWS

SIXTY-EIGHTH ANNUAL MEETING
Pittsburgh, Pa., October 17–20, 1939
HEADQUARTERS — HOTEL WILLIAM PENN

## CALENDAR OF THE 68TH ANNUAL MEETING

*Sunday, October 15*

9:00 A.M. Registration begins (Ball Room, William Penn Hotel).
9:30 A.M. Collegiate Council on Public Health Nursing—National Organization for Public Health Nursing.
10:00 A.M. Sixth Institute on Public Health Education opens.
2:30 P.M. Health Education Institute.
Collegiate Council on Public Health Nursing—National Organization for Public Health Nursing.
8:30 P.M. Community Mass Meeting—American Social Hygiene Association.

*Monday, October 16*

9:30 A.M. Pittsburgh Breakfast—under the auspices of the Women's Entertainment Committee. Everybody invited.
Association of Women in Public Health.
Conference of Municipal Public Health Engineers.
Conference of State Laboratory Directors.
Conference of State Sanitary Engineers.
International Society of Medical Health Officers.
Health Education Institute.
State Directors of Public Health Nursing.
Education Committee—National Organization for Public Health Nursing.
Luncheons.
Association of Women in Public Health.
International Society of Medical Health Officers and Health Education Institute.
2:30 P.M. American School Health Association.
Conference of Municipal Public Health Engineers.
Conference of State Laboratory Directors.
Conference of State Sanitary Engineers.
International Society of Medical Health Officers.
Education Committee—National Organization for Public Health Nursing.
State Directors of Public Health Nursing.
Health Education Institute.
4:00 P.M. Conference of Directors of Local Health Service.
4:30 P.M. Tea—Association of Women in Public Health.
Dinners. International Society of Medical Health Officers.
National Organization for Public Health Nursing.

*Tuesday, October 17*

9:15 A.M. Health Education Institute. Section Meetings.
2:30 P.M. Section Meetings.
8:30 P.M. Opening General Session.

*Wednesday, October 18*

9:15 A.M. Section Meetings.
11:45 A.M. General Session — Professional Education.
2:30 P.M. Section Meetings.
General Session—Medical Care.
Conference on Health Museums.
6:30 P.M. Pennsylvania Public Health Association and Tri-State Food and Health Officials Dinner.
8:30 P.M. Public Meeting.

*Thursday, October 19*

9:15 A.M. Section Meetings.
11:45 A.M. General Session—Cancer.
2:00 P.M. Sight-seeing Trip.
7:00 P.M. Annual Banquet.

*Friday, October 20*

9:15 A.M. Section Meetings.
2:30 P.M. Section Meetings.
5:00 P.M. Adjournment.
Late Evening. Departure of special trains for New York for delegates attending the New York World's Fair.

*Saturday, October 21*

NATIONAL HEALTH DAY at the New York World's Fair.

The abbreviated calendar above omits breakfast, luncheon and dinner sessions of sections, committees, and special groups. It also omits mention of many conferences of interest to limited numbers of delegates. No reference is made to the important and varied inspection trips. The final program, obtainable at the Registration Desk in the Ball Room of the William Penn Hotel, will convey full information about all these things.

Hotel and railroad rates have been published in the *Journal* as have also the summary of the scientific sessions and descriptions of places of public health interest in Pittsburgh which delegates will visit. Consult the July, August, and September issues. Come to Pittsburgh for the 68th Annual Meeting of your Association and meetings of related organizations.

## APPLICANTS FOR MEMBERSHIP

*The following individuals have applied for membership in the Association. They have requested affiliation with the sections indicated.*

### *Health Officers Section*

George W. Bassow, M.D., Main St., Woodstock, N. Y., Health Officer

Edwin H. Federman, M.D., Court House Annex, Eau Claire, Wis., City-County Health Officer

German Figueroa, M.D., C.P.H., Ayacucho 166, Barquisimeto, Venezuela, S. A., Health Officer

Erwin F. Hoffman, M.D., M.S.P.H., 805 Vulcan St., Iron Mountain, Mich., Director, Dickinson County Health Dept.

Joseph A. Johnson, M.D., Box 58, Greensboro, Ga., Commissioner of Health, Greene County Health Dept.

Raymond Luft, M.D., 1915 Elmwood Ave., Norwood, R. I., Superintendent of Health, City of Warwick

Harcourt A. Morgan, Jr., M.D., Athens, Tenn., Director, McMinn County Health Unit

Charles F. Nassau, M.D., LL.D., Sc.D., 503 City Hall Annex, Philadelphia, Pa., Director, Dept. of Health

Frederick P. Perkins, M.D., Capitol Annex, Room 103, Phoenix, Ariz., Superintendent of Public Health of Arizona

James E. Peterman, M.D., 624 Church St., Indiana, Pa., District Medical Health Officer

L. L. Sanford, M.D., University of Wyoming, Laramie, Wyo., Director of Student Health and University Physician

Maurice D. Vest, M.D., M.P.H., 619 Carson Ave., La Junta, Colo., Health Officer, Otero County Health Unit

### *Laboratory Section*

F. W. Janssen, 109 Grand St., Hoboken, N. J., Director of Laboratories, Janssen Dairy Corp.

Dr. Francesco N. Piccaluga, Blandengues 4350, Buenos Aires, Argentine, S. A., Director, Instituto Massone

Fries Shaffner, B.S. in Ed., 486 Avalon Rd., Winston-Salem, N. C., Serologist, Forsyth County Health Dept.

Jennie S. Siemienski, B.A., M.T., 10036 Jos. Campau, Hamtramck, Mich., Medical Technologist, Dept. of Health

### *Public Health Engineering Section*

Melvin A. Dobbs, B.S. in C.E., 325 East Broadway, East St. Louis, Ill., Sanitary Engineer, East Side Health District

Curtis B. Williams, B.S., State Dept. of Public Health, Santa Fe, N. Mex., State Milk Sanitarian

### *Child Hygiene Section*

Evelyn F. Buchheim, M.D., 682 Jean St., Oakland, Calif., Pediatrician, Bureau of Child Hygiene, State Health Dept.

Grace L. Dowell, R.N., 111 N. 3rd St., Arkansas City, Kans., Health Supervisor, Public Schools

Anita E. Faverman, M.D., 1838 Anza St.,

San Francisco, Calif., Pediatrician, State Health Dept.

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*Industrial Hygiene Section*

Marion F. Trice, B.S., 2004 Stone St., Raleigh, N. C., Industrial Hygienist, State Board of Health

*Food and Nutrition Section*

William V. Cruess, Ph.D., 339 Hilgard Hall, University of California, Berkeley, Calif., Assistant Professor in Food Technology

*Public Health Education Section*

Eleanore Aldworth, M.A., 2116 Devonshire Road, Ann Arbor, Mich., Hygiene Instructor, State Teachers College

Eva M. Bourne, R.N., 200 N. Michigan St., Roswell, N. Mex., City School Nurse

Paul Dodds, M.D., Room 510, South Office Bldg., State Capitol, Harrisburg, Pa., Director, Bureau of Maternal and Child Health, State Health Dept.

Donald A. Dukelow, M.D., 3744 46th Ave. S., Minneapolis, Minn., Director of Public Health Education, State Health Dept.

Alice L. Edwards, A.B., 306 State Bldg., Civic Center, San Francisco, Calif., Public Information Editor, State Health Dept.

Elizabeth C. Hoover, M.D., 1432 Potomac Ave., Dormont, Pa., Physician, Board of Health

Florence C. Kahn, D.H., 306 State Bldg., San Francisco, Calif., Dental Hygienist, State Health Dept.

Eloise Kloss, B.A., City Health Dept., Louisville, Ky., Director of Health Education

Meyer J. Plishner, B.S., M.P.H., Mill and Passaic Sts., Paterson, N. J., Public Relations Secretary, Passaic County Tuberculosis and Health Assn.

Ruth F. Rogers, B.A., Michigan Dept. of Health, Lansing, Mich., Consultant in Dental Health

Margaret M. Twombly, A.M., 1681 Shasta Ave., San Jose, Calif., Acting Head, Dept. of Health and Hygiene, San Jose State College

*Public Health Nursing Section*

Anna R. Ballo, 616 S. Third St., Raton, N. Mex., Colfax County Health Nurse

Jefferson I. Brown, R.N., State Board of Health, Phoenix, Ariz., Chief Nursing Consultant

Verna E. Hancock, Argonaut Hotel, Denver, Colo., General Field Supervisor, State Health Dept.

Winifred Humber, R.N., B.S., P. O. Box 233, Lone Pine, Calif., Public Health Nurse, Division of Child Hygiene, State Health Dept.

Sister Minalia, B.S. in Ed., R.N., St. Elizabeth Hospital, 49 Hopeland St., Dayton, Ohio, Director of Nurses

Marjorie J. D. McLean, 112 Riverside Ave., Decorah, Iowa, School Nurse, Board of Education

Catherine A. Sullivan, R.N., 318 S. 4th St., Steubenville, Ohio, City Health Nurse

Bessie Tattershall, R.N., 69 Main St., Tuckahoe, N. Y., Staff Nurse, Public Health Nursing Organization of Eastchester

Helen M. Wolfe, 305 State Bldg., San Francisco, Calif., Consultant in Orthopedic Nursing, State Health Dept.

Frances H. Ziegler, B.S., R.N., Vanderbilt University, Nashville, Tenn., Dean, School of Nursing

*Epidemiology Section*

John A. Carswell, M.D., D.P.H., P. O. Box 2658, Juneau, Alaska, Epidemiologist, Dept. of Health

Richard J. Kettering, City Health Office, Aberdeen, S. D., Milk Sanitarian

Charles H. Mann, Jr., M.D., M.S.P.H., 301 N. Erie St., Bay City, Mich, Assistant, Division of Hygiene and Public Health, University of Michigan

James M. Mather, M.D., D.P.H., 19 Grand St., Coldwater, Mich., Assistant to Branch County Health Director

*Unaffiliated*

R. H. Lucas, 925 Main St., Columbia, S. C., Accountant, State Board of Health

Frances A. Macdonald, A.B., 236 Bay State Road, Boston, Mass., Junior Epidemiologist, State Dept. of Health

Cyrus H. Maxwell, M.D., 3 Winding Road, Elsmere, N. Y., Chief, Health Service Bureau, State Education Dept.

Charles E. McBrayer, M.D. (Lieut. Col. U.S.A., Retired), Hotel Multnomah, Portland, Ore.

Galina Mouromseff, B.A., 219 Montclair Ave., Montclair, N. J., Research Dept., Westinghouse Electric and Manufacturing Co.

Lucy V. Reardon, M.A., National Institute of Health, Washington, D. C., Associate Protozoölogist

# APPLICANTS FOR FELLOWSHIP

(Additional to names published in August and September *Journals*)

*In accordance with the By-laws of the Association, the names of applicants for Fellowship are officially published herewith. They have requested affiliation with the Sections indicated. Action by the various Section Councils, the Committee on Eligibility, and the Governing Council will take place between now and the time of the Pittsburgh Annual Meeting.*

*Health Officers Section*

William H. Best, M.D., Deputy Commissioner, New York City Dept. of Health, New York, N. Y.

Regnar M. Sorenson, M.D., C.P.H., Director, Division of Venereal Disease Control, State Dept. of Health, Des Moines, Iowa

Maysil M. Williams, M.D., C.P.H., State Health Officer, Bismarck, N. D.

*Laboratory Section*

N. Paul Hudson, M.D., Ph.D., Professor and Chairman, Dept. of Bacteriology, Ohio State University, Columbus, Ohio

Murrell O. Robinson, V.M.D., Professor of Bacteriology, Alabama Polytechnic Institute, Auburn, Ala.

Fred D. Stimpert, Ph.D., Medical Microbiologist, Los Angeles County Hospital, Los Angeles, Calif.

*Public Health Engineering Section*

Milton H. Bidwell, B.S., Bacteriologist, New York State Conservation Dept., Oceanside, L. I., N. Y.

Francis M. Dawson, M.C.E., Dean, College of Engineering, University of Iowa, Iowa City, Iowa

*Industrial Hygiene Section*

Edward G. Meiter, Ph.D., Director, Industrial Hygiene Laboratory, Employers Mutual Liability Insurance Co., Milwaukee, Wis.

*Food and Nutrition Section*

Frederic W. Nordsiek, S.B., Associate, Dept. of Nutrition, American Institute of Baking, New York, N. Y.

*Child Hygiene Section*

Francis V. Corrigan, M.D., Chief, Division of Maternal and Child Health, State Dept. of Health, Providence, R. I.

*Public Health Education Section*

Harold S. Diehl, M.D., Sc.D., Dean of Medical Sciences, University of Minnesota, Minneapolis, Minn.

Bess Exton, M.A., C.P.H., Assistant Health Educator, National Education Assoc., Washington, D. C.

D. Clare Gates, Dr.P.H., M.S.P.H., Director of Health Education, State Dept. of Health, Bismarck, N. D.

Ruth E. Grout, C.P.H., Ph.D., Senior Supervisor of Health Education, Dept. of Health and Safety, Tennessee Valley Authority, Chattanooga, Tenn.

Leo H. Mynes, M.D., Medical Director, Kanawha County School Health Service, Charleston, W. Va.

Delbert Oberteuffer, Ph.D., Professor of Physical Education and Chairman, Division for Men, Ohio State University, Columbus, Ohio

Anna B. Towse, B.S., Executive Secretary, Good Teeth Council for Children, Inc., Chicago, Ill.

Margaret H. Tracy, A.B., Executive Secretary, Boston Health League, Boston, Mass.

David B. Treat, A.M., Director of Public Health Education, Dept. of Public Health, Flint, Mich.

Kathleen W. Wootten, M.A., Head, Health Dept., Georgia State College for Women, Milledgeville, Ga.

*Epidemiology Section*

Harold W. Brown, M.D., Dr.P.H., Professor of Public Health, University of North Carolina, Chapel Hill, N. C.

Bernard M. Blum, M.D., M.P.H., Assistant District State Health Officer, State Dept. of Health, Buffalo, N. Y.

*Unaffiliated*

Robert P. Fischelis, Phar.D., B.Sc., Secretary and Chief Chemist, Board of Pharmacy of the State of New Jersey, Trenton, N. J.

Marie C. Harrington, M.A., Educational Director, Dairy Council of St. Louis, St. Louis, Mo.

Bruce H. Pollock, M.D., C.P.H., District Health Officer, State Dept. of Health, Point Pleasant, W. Va.

## THE WESTERN BRANCH REPORTS ON TEN YEARS OF PROGRESS

FROM the Western Branch of the A.P.H.A. comes an interesting and illuminating report of ten years of progress as recorded by Dr. William P. Shepard who retired as Secretary of the Western Branch at its Oakland meeting in July. Dr. Shepard pays tribute for remarkable progress to the vigorous and courageous national leadership in the Public Health Service, and to the principles for which the American Public Health Association and the Western Branch have always stood, namely, an abiding faith in the importance and values of the public health cause and a willingness to work together for public health advancement regardless of personal glory.

The Western Branch has reached its Tenth Annual Meeting with a membership of 1,437. Of this number 127 are Fellows and 871 active members of the A.P.H.A., and 439 are regional members. In 10 years the A.P.H.A. membership in the Western Branch has increased from 368 to 998—an increase of over 170 per cent! It is interesting to note also that the A.P.H.A. constituency in the Western Branch represents 15 per cent of the total A.P.H.A. membership. Since the last Branch meeting there has been a gain of 197 members, and not one state, province, or territory shows a loss in membership.

In 1929 when the Branch was organized, only 34 counties in its territory had full-time units. Today there are 64 counties with full-time health units and 75 more counties served by full-time health districts—a total of 139. Similarly, in 1929 there were 220 full-time employees engaged in health service in 32 counties (excluding Los Angeles and San Francisco). This year there are 825 full-time and 46 part-time public health workers engaged in the 137 counties. Ten years ago there were 293 full-time employees in the 11 state health departments comprising Western Branch territory. This year there are 513, an increase of 220 or 75 per cent. Combining state and county full-time personnel, there were 513 in 1929, and in 1939 there are 1,338, an increase of 825 or 161 per cent. Unfortunately figures for the Provinces of Canada, the cities and Territories of the Western Branch are not available but there can be no doubt that here too the story would be as encouraging.

The influence of western public health workers in the affairs of the parent body is evidenced by the fact that today Western Branch members hold 53 A.P.H.A. offices or committee memberships. In 1930, at the time of the First Annual Meeting there were 30 such incumbents. The interest of Western Branch members in national health affairs is further evidenced by the yearly increase in their attendance at A.P.H.A. Annual Meetings.

When the Western Branch was organized, there was one Western state public health society affiliated with the A.P.H.A. Today there are 6 with an application pending from one state and another expected shortly.

Canadian members of the A.P.H.A. have been most enthusiastic in their support of the Western Branch and faithful in their attendance at its meetings. The Canadian membership has increased from 7 to 31.

The only two remaining Territories of the United States are included in the Western Branch. Through the efforts of the late Dr. F. E. Trotter, the membership in Hawaii has increased from 0 to 31. Dr. W. W. Council of Alaska is responsible for the increase in Alaska from 0 to 22.

Direct public health activities of the

Western Branch are a matter of record. They loom large when the scientific papers presented at annual meetings, the minutes, committee reports, and resolutions are surveyed. Among them are:

Early recognition of the health hazards connected with the construction of Boulder Dam and recommendation to the proper persons that these be given careful study. The health record of this enormous undertaking was surprisingly good, and the importance of preventive medicine in industry was once more demonstrated.

Repeated pleas for national assistance with the health problems of migratory workers, especially with tuberculosis. Only in recent years have these pleas been heeded.

Recognizing the value and helping to stabilize research in the control of Rocky Mountain Spotted Fever. Attention has been directed to the general prevalence of the disease, its eastward spread, and the necessity of ample supplies of vaccine. The Laboratory at Hamilton, Mont., has been assumed by the U. S. Public Health Service, additional buildings provided, and the staff increased.

Attention of state and national legislators has been repeatedly called to the importance of maintaining adequate funds for health protection and for providing trained personnel.

Urging the establishment of a western training school for public health personnel. Three years ago such a school was established at the University of California. Nearly 50 health officers have been trained as well as over 100 other individuals.

Public recognition in 1935 of the fact that plague infection was widespread among various rodents of the West. Immediate and intensive study was called for. The term "Sylvatic Plague" was introduced by Dr. Karl F. Meyer to distinguish this endemic wild rodent disease from epidemics of plague customarily associated with domestic rodents and man. Annual reports have been made and published since.

Appointment of a committee to promulgate and encourage newer methods of diagnosis and treatment of lobar pneumonia.

Equally interesting and important activities have been the discovery of "Valley Fever" and its relation to coccidioidal granuloma, and control of botulism and mussel poisoning. There are others too numerous to mention.

The original object of the Western Branch as stated in its Constitution is "to protect and promote public and personal health, and to promote better health service in the Western portions of the United States and the Dominion of Canada." In the 11 years of its existence the Western Branch has demonstrated its usefulness and the worthiness of this original object. The Western Branch has had virtually no money except a modest sum for limited mailing and printing from the parent organization. It has had no full-time personnel. Its work has been done by voluntary officers and committees.

The experiences recorded here have contributed to the success of this unique organization. They are made part of the record in order that they may not be lost sight of in the future.

### CHARLES H. HOOD EDUCATIONAL TRUST

THE Charles H. Hood Educational Trust of Boston has made a grant of $6,000 for the work of the American Public Health Association's Committee on Coördination in Health Education (Joint Committee between the Health Officers and Public Health Education Sections). Professor C. E. Turner of the Massachusetts Institute of Technology is Chairman.

The grant will be administered through the Health Education Research Laboratory at the Massachusetts Institute of Technology.

### NEWS FROM THE SOUTHERN BRANCH

THE Southern Branch of the American Public Health Association will meet in Memphis, Tenn., during the annual sessions of the Southern Medical Association, November 21–24. The Branch meetings will be held on Tuesday and Wednesday, November 21–22.

Dr. P. E. Blackerby, Assistant State Health Commissioner of Kentucky, who is Secretary of the Southern Branch, has announced that with the approval of President J. N. Baker there have been made the following changes in the Governing Council and the Regional Executive Board of the Branch. Dr. James A. Hayne, Columbia, S. C., succeeds Dr. J. N. Baker as a member of the Regional Executive Board; and Dr. Felix J. Underwood, Jackson, Miss., is reappointed as a member of this Board. The following persons are also appointed to the Governing Council of the Southern Branch: Dr. Warren F. Draper, District of Columbia; Dr. H. F. Parker, Jefferson City, Mo.; Dr. W. C. Williams, Nashville, Tenn., and Dr. I. C. Riggin, Richmond, Va.

### SUBSCRIPTION AGENT IMPOSTER

IT has been reported that a WILLIAM DALTON has been offering cut rate prices in Texas on subscriptions to the *American Journal of Public Health, The Trained Nurse and Hospital Review, Modern Hospital,* and other publications. It is stated that he has collected money, giving receipts bearing the name "National Circulation Co., Inc., 343 N. Michigan Blvd., Chicago, Ill." Efforts to reach the National Circulation Company at this address have failed.

Members of the Association are again warned that no solicitors are employed by the *American Journal of Public Health.* Subscriptions should be placed direct with the Association office or through established subscription agencies.

### DECEASED MEMBERS

Ernest C. Dickson, M.D., San Francisco, Calif., Elected Member 1920, Elected Fellow 1934

Lucius W. Holloman, M.D., Marksville, La., Elected Member 1936

Lloyd A. Masterson, M.D., New Orleans, La., Elected Member 1936

Wilbur A. McPhaul, M.D., Jacksonville, Fla., Elected Member 1936

F. E. Trotter, M.D., Honolulu, Hawaii, Elected Member 1924

# EMPLOYMENT SERVICE

The Employment Service will register persons qualified in the public health field without charge.

Replies to these advertisements, when keyed, should be addressed to the American Public Health Association, 50 West 50th Street, New York, N. Y., identifying clearly the key number on the envelope.

## Positions Wanted

### HEALTH OFFICERS

Experienced physician, administrator, epidemiologist and teacher, now employed, with C.P.H. from Johns Hopkins, and 14 years' public health background, will consider position. Prefers epidemiology in city or state department. Excellent references. **A355**

Experienced physician who has been local health officer and school physician in New York State, now has M.P.H. from Harvard School of Public Health, and wishes position in administration, epidemiology, or public medical service. **A423**

Physician, M.D., Yale; M.S. in field of public health, Columbia; also short course for health officers, Vanderbilt; good clinical background, 14 months' public health experience; will consider appointment in child health, epidemiology, or public health administration. **A350**

Physician, M.D., University of Cincinnati; with postgraduate training in venereal disease control, Johns Hopkins; is available as venereal disease control officer. **A363**

Physician, experienced in health administration of cities and states, will consider attractive opening in maternal and child health or health education. **A343**

Physician, M.D., Vanderbilt University; Dr.P.H., Johns Hopkins; experienced as epidemiologist, health officer and professor of preventive medicine, seeks position as administrator or epidemiologist. **A397**

Physician, M.P.H., Harvard; well experienced in city and rural health administration, will consider appointment as district health officer or in city or state health department. **A418**

Well qualified physician, M.D., Rush; M.S.P.H., University of Michigan; with 3 years' residence in tuberculosis, and special interest in venereal disease control, seeks responsible appointment. Excellent references. **A406**

Physician, M.D., University of Cincinnati; M.P.H., Harvard; postgraduate studies in bacteriology and immunology, will consider position as health officer or epidemiologist. Has served as director of county health unit, director of industrial hygiene and medical statistics and venereal disease field survey officer. **A305**

Physician, aged 33; M.D., Minnesota; Dr.P.H., Johns Hopkins; has served as epidemiologist in large city and director of county health unit, with 7 years' public health experience; will consider position in public health administration or epidemiology. **A432**

Physician, M.D., Class A medical school; completed short course for health officers at Vanderbilt; experienced health officer and director of county health unit, will consider appointment as health officer or school physician. **A320**

Physician, M.D., Columbia; experienced as county health officer and superintendent of health, seeks position in public health administration or medical executive work. **A396**

Physician, M.D., Johns Hopkins; M.S.P.H., University of Michigan; experienced in school and city health work; for the past year director of county health department, seeks position as health director of city school system or in health service of a college or university. Will also consider position as epidemiologist or health officer in urban health department. **A354**

### HEALTH EDUCATION

Young woman, experienced teacher in health education, with M.S. in Public Health and Hygiene from University of Michigan, seeks position as health coördinator. **H398**

Well qualified woman in health education wishes position as health coördinator or health counselor. Has wide experience, and Ph.D. from New York University. **H236**

Young woman, Ph.D., Columbia Uni-

versity, splendid background of experience in health education, will consider position as director of public health education. **H294**

Young woman, M.A., Health Education, Teachers College, Columbia; with splendid international experience, seeks position as director of health education. **H369**

Young man, M.S.P.H., University of Michigan, experienced in an eastern state department of health and as a university instructor, desires position in popular health instruction, especially by written media, with official or volunteer health agency or with commercial enterprise, insurance company or editorial work. **H435**

### LABORATORY

Capable research worker, Ph.D., trained at University of Southern California and Pasteur Institute, seeks position directing laboratory, in research work or field investigation. Has taught bacteriology, directed state hygiene laboratory and hospital laboratories. **L315**

Broadly experienced laboratory director with excellent background, now employed southern state, seeks responsible position in laboratory work or teaching of bacteriology, immunology, or pathology. **L426**

Physician, C.P.H., Harvard-Technology; experienced as bacteriologist and pathologist and director of state laboratories, desires position. **L208**

Bacteriologist, M.S.P.H., University of Michigan; 2½ years as serologist in private hospital; desires work as serologist or bacteriologist in public health laboratory. Willing to start modestly. **L425**

Woman bacteriologist; B.A., Chemistry, 1933, 5 years' experience in experimental and practical therapeutics including animal work; desires position in public health field. **L394**

Young man bacteriologist, aged 33, with training in serology; for the past 4 years bacteriologist in charge, public health laboratory; will consider opening. **L427**

Chemist and bacteriologist experienced in water supply, sewage, and public health laboratory work. Five years' experience. B.S., 1933. Desires work in sanitation or general public health. **L412**

Chemist and bacteriologist, university graduate, M.A. and M.S., with 15 years' experience in different phases of public health work, desires position. **L438**

Experienced woman bacteriologist, Ph.D., University of Illinois, 1937, wishes position in teaching or research. Excellent bibliography and references. **L410**

Young woman, registered medical technologist with 10 years' experience, specializing in the isolation of typhoid carriers, also trained and experienced in medical social work, desires position in public health laboratory. Prefers South. **L438**

### MISCELLANEOUS

Physician, M.D., class A medical school; training in obstetrics and public health. Experienced as director of county health department, teacher of clinical obstetrics and administrator of state maternal and child health program, desires position in public health obstetrics or in maternal and child health administration. **C417**

Dentist, graduate of Temple University, with excellent postgraduate experience, desires position in administrative aspects of dental hygiene. **M352**

Experienced teacher in bacteriology and public health; Ph.D., Cornell; now professor in grade A medical school, will consider teaching, executive or administrative position. **M327**

Engineer with good training and experience in water treatment, sewage plant operation and in research, wishes position as superintendent. Can go anywhere. **E422**

Experienced director of milk control, food and sanitation and industrial hygiene. B.S. degree and well known record directing such work in two major cities and one state. References prominent public health personalities. **I 434**

Engineer, sanitary inspector, lecturer, long experience in large city department of health, will undertake a new assignment in above fields or in administration. Warm climate preferred. **M436**

Physician, 32, married, M.D., 1936; postgraduate course in v.d. control, experienced as district health officer and in organizing and publicizing syphilis control campaign; now employed in charge venereal disease clinics in metropolitan health department; seeks v.d. control post with emphasis on organizational, investigative-survey or administration. **M437**

# Situations Open

PUBLIC HEALTH PHYSICIAN--Young physician with good medical background, interested in obtaining public health training; salaried appointment at end of month in field; man of English, Irish or Scotch ancestry preferred. 100-PH, Medical Bureau, Palmolive Building, Chicago.

PUBLIC HEALTH PHYSICIAN—To be sponsored jointly by state department of health, Protestant mission; rural district; woman physician considered; $2,700; South. 101-PH, Medical Bureau, Palmolive Building, Chicago.

STUDENT HEALTH PHYSICIANS—(a) Exclusive junior college for women; man or woman with Master's or Ph.D. degree, primarily trained internal medicine. (b) Co-educational college; man under 40 required; $250 monthly, 11 months; Illinois. (c) Eastern university; recent graduate eligible; September–June; $150. 102-PH, Medical Bureau, Palmolive Building, Chicago.

SCHOOL PHYSICIAN—Recent graduate contemplating public school health service as life work; duties include some teaching and dispensary service for students; eastern university; $200. 103-PH, Medical Bureau, Palmolive Building, Chicago.

INDUSTRIAL NURSE—Registered nurse capable assuming complete charge industrial first-aid station; southwest; $150, maintenance. 104-PH, Medical Bureau, Palmolive Building, Chicago.

STUDENT HEALTH NURSE—Graduate nurse, trained public health work, for student health staff state normal school; 105-PH, Medical Bureau, Palmolive Building, Chicago.

PUBLIC HEALTH NURSE—Graduate nurse with both public health and tuberculosis experience; busy tuberculosis follow-up service, city of 40,000; $115, increasing, plus car allowance; North. 106-PH, Medical Bureau, Palmolive Building, Chicago.

STUDENT HEALTH NURSE—Duties largely general duty in infirmary exclusive college for women; 2 years' college training required; $100, meals. 107-PH, Medical Bureau, Palmolive Building, Chicago.

ASSISTANT SCHOOL NURSE—Public school system, city of 10,000; graduate nurse trained for public health certificate required; $1,100, nine months; midwest. 108-PH, Medical Bureau, Palmolive Building, Chicago.

LABORATORY TECHNICIAN—Preferably one with public health laboratory experience; should be exceptionally trained bacteriology and serology; excellent future for right person. 109-PH, Medical Bureau, Palmolive Building, Chicago.

# Situations Wanted

PUBLIC HEALTH PHYSICIAN—A.B., midwestern college; M.D., Johns Hopkins; M.S.P.H., University of Michigan; interesting record of successful experience including long term as school physician in fashionable suburb; for further information, please write M. Burneice Larson, Director, Medical Bureau, Palmolive Building, Chicago.

BACTERIOLOGIST — B.A., eastern school; C.P.H. and Ph.D. (bacteriology), Yale University; has done considerable research; 4 years, bacteriologist and research technician, public health laboratories; desires appointment in public health laboratories; for further details please write M. Burneice Larson, Director, Medical Bureau, Palmolive Building, Chicago.

HEALTH EDUCATOR—A.B., Wellesley; M.S. in public health from state university; 6 years, teaching physiology and sciences in public schools; 2 years, director of child health education, national organization; 2 years, health counselor and teacher of health education, midwest metropolis; 4 years' social work; for further details, please write M. Burneice Larson, Director, Medical Bureau, Palmolive Building, Chicago.

PUBLIC HEALTH NURSE—Graduate of a fairly large training school of nursing; graduate training in public health nursing at the University of Minnesota; 10 years' experience as public school nurse; for further details, please write M. Burneice Larson, Director, Medical Bureau, Palmolive Building, Chicago.

# NEWS FROM THE FIELD

## 1939 EDITION OF CONNECTICUT OFFICIAL MILK SEDIMENT STANDARDS

THE State Dairy and Food Commissioner and the State Commissioner of Health have approved the new 1939 Edition of the Connecticut Official Milk Sediment Standards. This standard is the only one acceptable and approved for use in laboratories making milk tests in Connecticut, and to obtain better uniformity of grading milk sediment it is urged it be used for sediment testing done in the field and at milk plants. The new standard has been specially adapted to field and plant use.

In the new edition the seldom used "Excessively Dirty" discs have been eliminated and the standard carries only three sediment discs which grade milk into four cleanliness ratings; namely, Clean, Good, Poor, and Dirty. The amounts of sediment on the pads of the new standard correspond roughly with sediment scores of 13, 50, and 100 on the 1931 Edition.

Public health laboratories in Connecticut approved for the examination of milk have been requested by the State Commissioner of Health to begin use of the 1939 Edition as soon as possible and instructed to have it in use not later than December 31, 1939. Notification has been sent out that the 1931 Edition should not be used after December 31, 1939.

For many years the Connecticut Official Milk Standard has been used extensively outside of Connecticut for the grading of milk both in the United States and in other countries. In *Standard Methods for the Examination of Dairy Products,* Seventh Edition, 1939, reference is made to photographic standards and those who do not wish to prepare their own discs are referred to the copies of the photographic standards prepared by the Connecticut State Department of Health Laboratory, Hartford. The report states:

> The standards on this photograph are based on pint samples of milk to which weighed amounts of sediment have been added. The 1931 standards cover the range from clean to very dirty milk with numerical ratings or sediment scores given for each disc. The 1939 standards have been simplified by the elimination of cleanliness ratings for dirty milk.

Persons outside of Connecticut placing orders for milk sediment standards should state whether the new 1939 Edition or the old 1931 Edition of the standard is desired. The price of the new 1939 Edition is sixty cents, but the price of the old 1931 Edition will remain $1.00 a copy for the few persons who may wish to continue using the old standard.

## THE FIRST ANNUAL SYMPOSIUM FOR INDUSTRIAL NURSES

THE Illinois State Department of Public Health, through its Divisions of Industrial Hygiene and Child Hygiene and Public Health Nursing, has arranged a symposium for industrial nurses in the state, to be held October 26–28, at the University of Illinois Medical School, in Chicago.

Coöperating organizations will be: Chicago Industrial Nurses Association, Greater Chicago Safety Council, Illinois Manufacturers' Association, American Industrial Hygiene Association, American Medical Association, and University of Illinois Medical School.

## UNIVERSITY OF CALIFORNIA NURSING EDUCATION INSTITUTE

AS part of the Summer Session program of the University of California, the Division of Nursing Education of the Department of Hygiene held

a 3 weeks' Institute on "Social and Nursing Problems in the Control of Syphilis and Gonorrhea." Mrs. Evangeline Morris, of Simmons College, was the prime moving force of the Institute. Dr. Malcolm Merril, Chief of the Bureau of Venereal Diseases, California State Department of Public Health, gave a well organized practical view of the medical aspects in the control of syphilis and gonorrhea.

Enrollment included public health nurses from state, city, and county health departments, Indian Service schools, Visiting Nurse Associations, American Red Cross Nursing Services, and nurses from clinic, general duty, and private duty fields.

### TRI-STATE FOOD AND HEALTH OFFICIALS CONFERENCE

THE Tri-State Food and Health Officials Conference will meet at Pittsburgh on Wednesday, October 18, during the Annual Meeting of the A.P.H.A. This Conference is composed of health and milk control officials in the States of Ohio, West Virginia, and Pennsylvania. The officers are:

*President:* Julius A. Pizzoferrato
*Secretary-Treasurer:* Thomas Henderson
*First Vice-President:* Dr. Reese Pedicord
*Second Vice-President:* Elmer Harter
*Editor:* Leicester Patton

Their program will include papers by Dr. W. W. McFarland, President, Pennsylvania Public Health Association; Dr. Reese Pedicord, Wheeling, W. Va.; and Fred L. Schoenberger, Secretary, Ohio Dairy Products Association, Columbus, Ohio.

### FILTERABLE VIRUSES RESEARCH

A NEW laboratory for the study of filterable viruses has been established at New Brunswick, N. J., by the Squibb Biological Laboratories. A special building has been equipped for the research.

Raymond C. Parker, Ph.D., a member of the staff of the Rockefeller Institute for Medical Research, has been appointed Director.

## PERSONALS

### Central States

FORREST J. AUSTIN, M.D.,† formerly of Houghton, Mich., has been appointed Director of a district health unit for Webster, Calhoun, and Boone Counties, Iowa.

DR. HARRISON C. BLANKMEYER has been appointed Superintendent of Health of Springfield, Ill.

DR. GEORGE W. BOWMAN, of Indianapolis, Ind., has been appointed State Director for Venereal Disease Control in the Indiana State Health Department, succeeding DR. WENDELL C. KELLY, resigned.

DR. ADELBERT R. CHAMBERS, of Iola, Kans., has been appointed Health Officer of Allen County.

DR. HOWARD F. CRAIG, of Protection, Kans., has recently been appointed Health Officer of Comanche County.

DR. MAX L. DURFEE, of the staff of the Health Service of the University of Michigan, Ann Arbor, has been appointed Director of the Health Service at Iowa State Teachers College, Cedar Falls, Iowa.

MARIE A. HAGELE, M.D.,† of Chicago, Ill., has been appointed Regional Consultant in Maternal and Child Health for the Michigan State Department of Health.

KALMAN S. VON HAITINGER, M.D.,† formerly of the Hillsdale County, Mich., Health Department, has been appointed Health Officer of the Midland County Health Department, succeeding EDWIN H. PLACE, M.D.,† resigned.

ERWIN F. HOFFMAN, M.D., M.S.P.H.,† of Mason, Mich., has been appointed Director of the Dickinson County Health Unit, to succeed PHILIP E. M.

† Member A.P.H.A.

BOURLAND, M.D., M.S.P.H.,† of Iron Mountain, resigned.

DR. WENDELL C. KELLY, of Indianapolis, Ind., has resigned as State Director for Venereal Disease Control in the Indiana State Health Department, to become associated with Eli Lilly and Company.

DR. FLOYD H. KINYOUN has resigned as Health Officer of Omaha, Nebr., to enter private practice in Los Angeles, Calif.

DR. HERBERT R. SCHMIDT, of Newton, Kans., has been appointed Health Officer of Harvey County, to fill the unexpired term of the late DR. WILLIAM F. SCHROEDER.

DR. ELLIS K. VAUBEL, of Indianapolis, Ind., has been appointed Assistant Director of the Division of Preventable Diseases of the Iowa State Department of Health, succeeding DR. PAUL STEPHEN, who resigned to enter private practice at Manchester, Iowa.

### Eastern States

CLIFFORD W. BEERS, of New York, founder of the mental hygiene movement, has resigned as Secretary of the National Committee for Mental Hygiene, after more than 30 years of service. He will continue his work as Secretary of the American Foundation for Mental Hygiene, and the International Committee for Mental Hygiene.

DR. JOHN L. BOND, of Lehighton, Pa., has been appointed Medical Director of Carbon County.

WALTER CLARKE, M.D.,† Executive Director of the American Social Hygiene Association, New York, N. Y., has been awarded the rank of Knight of the Legion of Honor by President Lebrun of France. The citation was made for Dr. Clarke's work with the League of Red Cross Societies and other international health activities in which the French Government is interested.

JOHN B. GRANT, M.D., C.P.H.,* of the staff of the International Health Division, Rockefeller Foundation, who for more than 20 years has been assigned to China, has been appointed by the Government of India as Director of the All India Institute of Hygiene and Public Health and, concurrently, Professor of Public Health Administration. Dr. Grant arrived in Calcutta late in June to begin his responsibilities.

DR. MILTON E. KIRKPATRICK, of New York, recently Director of a child guidance center at Lansing, Mich., has been appointed Director of the Division on Community Clinics, of the National Committee for Mental Hygiene, New York, N. Y. He succeeds DR. GEORGE S. STEVENSON, who recently became Medical Director of the National Committee for Mental Hygiene.

DR. GEORGE A. MARSDEN, of Oswego, N. Y., has been appointed City Health Officer, to succeed the late DR. JAMES E. MANSFIELD.

ROSCOE L. MITCHELL, M.D.,† Assistant Director of the Bureau of Health in the Maine State Department of Health and Welfare, has been appointed Director, to succeed GEORGE H. COOMBS, M.D.†

DR. JAMES E. PETERMAN, M.D.,† of Cherry Tree, Pa., has been appointed District Medical Director of Indiana, Armstrong, and Jefferson Counties.

DR. ANTHONY J. SPARTA, of Easton, Pa., has been appointed Medical Director of Northampton County, succeeding DR. FRANK J. CONAHAN, of Bethlehem.

HOWARD CANNING TAYLOR, JR., M.D., of New York, has been appointed to the William Goodell Professorship at

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* Fellow A.P.H.A.
† Member A.P.H.A.

the University of Pennsylvania School of Medicine.

KATHRYN C. TRENT,† of Dover, Del., has resigned as Director of the Division of Public Health Nursing for the Delaware State Board of Health, which position she has held for the past 4 years, to accept a position with the Birth Control Federation of America as a Supervisor of the Southern Region. Her southern address is not yet announced.

ALLAN A. TWICHELL,† of New Haven, Conn., Technical Secretary of the American Public Health Association's Committee on the Hygiene of Housing, lectured at the Housing Management Training Institute held at New York University, July 24–August 18. His subject was "Current Thoughts on the Relation of Housing to Health, Delinquency and Other Major Social Problems." The Institute was conducted in coöperation with the National Association of Housing Officials and with the Assistance of Staff members of the United States Housing Authority.

DR. MATTHEW P. WARD, of Brownsville, Pa., has been appointed Medical Director of Fayette County, to succeed DR. J. GLENN HEMINGTON, of Uniontown.

J. M. WISAN, D.D.S.,* of Elizabeth, N. J., has been appointed Consultant to the Dental Health Education Program of the New Jersey State Department of Health. Dr. Wisan was chairman of the Council on Mouth Hygiene of the New Jersey State Dental Society for a number of years, and during the past year he has served as Secretary of the Oral Health Group, American Public Health Association.

## Southern States

DR. FLETCHER R. ADAMS, of Monroe, N. C., Health Officer of Union County, has been appointed Health Officer of Catawba County, to succeed DR. CLARENCE H. WHITE, formerly of Newton, N. C., and now of Philadelphia, Pa.

DR. NEALE M. ATKINS, of Greenville, Ky., Health Officer of Muhlenberg County, has been transferred to Graves County, to take charge of a new unit with headquarters at Mayfield. DR. JAMES R. BARCUS, of Gladewater, Tex., has been appointed Health Officer of the town.

DR. RICHARD B. AUSTIN, of Forest, Miss., has been named part-time Health Officer in Scott County, filling the unexpired term of DR. WILLIAM E. ANDERSON.

DR. LOREN V. BAKER, of Elk City, Okla., has been appointed Health Officer of Beckham County.

DR. JOSHUA P. BEAM, of Arnett, Okla., has been appointed Health Officer of Ellis County.

DR. WILLIAM PRICE BITTINGER, of Summerlee, W. Va., has been appointed a member of the Public Health Council, succeeding the late DR. SAMUEL W. PRICE, of Scarbro.

DR. WILLIAM S. CARY, of Reydon, Okla., has been appointed Health Officer of Roger Mills County.

DR. GEORGE R. CREEKMORE, of Brooksville, Fla., has been appointed a member of the Board of Commissioners of Hernando County.

DR. JOHN I. DERR, of Waurika, Okla., has been appointed Health Officer of Jefferson County.

DR. DONALD W. DYKSTRA, of Morrilton, Ark., has been appointed in charge of the new Syphilis Control Division of the Arkansas State Department of Health. He will assist ARTHUR M. WASHBURN, B.S., M.D.,† of Little Rock, Director of the Division of Communicable Disease

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† Member A.P.H.A.

Control. Another member of the new Division will be ARTHUR B. PRICE, M.D., M.P.H., of the U. S. Public Health Service, who has been assigned to the State Department for a year of field training.

DR. PAUL T. ERICKSON, of the U. S. Public Health Service, arrived in Jackson, Miss., recently to launch a year's program in the control of venereal diseases, in coöperation with the Mississippi State Department of Health.

DR. FRANCIS POLK FRY, JR., of Frederick, Okla., has been appointed Health Officer of Tillman County.

DR. SIMON S. GARRETT, of Duncan, Okla., has been appointed Health Officer of Stephens County.

DR. ELLERY C. GAY, of Little Rock, Ark., has been appointed Medical Supervisor of the Crippled Children's Division of the Arkansas State Department of Public Welfare, serving on a part-time basis. He succeeds DR. LEE VALLETTE PARMLEY, resigned.

PAUL M. GOLLEY, M.D.,† of Chapel Hill, N. C., has been appointed Health Commissioner of Walker and Catoosa Counties, Ga., succeeding DR. RICHARD C. SHEPARD, resigned.

DR. WILLIAM F. GRIFFIN, of Watonga, Okla., has been appointed Health Officer of Blaine County.

WILLIAM GROSSMAN, M.D.,† Epidemiologist of the Virginia State Health Department, Richmond, Va., has been appointed Director of the Bureau of Communicable Diseases, succeeding G. FOARD MCGINNES, M.D.*

DR. FERDINAND R. HASSLER, JR., of Seminole, Okla., has been appointed Health Officer of Pottawatomie County.

DR. DON V. HATTON, of Leitchfield, Ky., has resigned as Health Officer of Grayson County, to take a similar position in Virginia.

EDWARD M. HOLMES, JR., M.D.,† formerly Health Officer of Fairfax County, Va., has been appointed Director of the Division of Venereal Disease Control of the Virginia State Department of Health, Richmond, Va.

DR. JOHN B. HOZIER, of the U. S. Public Health Service, has been detailed to the West Virginia State Department of Health, at Charleston, as Acting Director of the Bureau of Venereal Disease, in the absence of DR. CHARLES N. SCOTT, who will spend a year at Johns Hopkins University.

DR. CHARLES F. HULL, of Carthage, Tex., has been appointed Health Officer of Panola County.

DR. MELVIN L. HUTCHESON, of Denton, Tex., has been appointed Health Officer of Denton County.

DR. WALTER R. JOBLIN, of Porter, Okla., has been appointed Health Officer of Wagner County.

RANSOM J. JONES, M.D.,† of Poplarville, Miss., formerly Director of the Pearl River County Health Department, has been named to a similar position in the southeastern district which was organized in March, composed of George, Greene, Perry, and Stone Counties; DR. JOHN W. DUGGER, of Jackson, has been acting head.

DR. LLOYD T. LANCASTER, of Cherokee, Okla., has been appointed Health Officer of Alfalfa County.

DR. BERT H. MALONE has been appointed Regional Medical Director of the Southeastern Regional Office, with headquarters in Waycross, Ga. This is one of the six regional health units that have been established under the supervision of the Georgia State Department of Health in an expanded state health program.

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† Member A.P.H.A.

Other new directors are:

Dr. Robert B. Griffin, Northwestern District, Marietta, Ga.

Dr. Aaron Wilson Brown, Northeastern District, Gainesville, Ga.

Rufus Floyd Payne, M.D.,* West Central District, Griffin, Ga.

Dr. John M. Walton, East Central District, Swainsboro, Ga.

Lester M. Petrie, M.D.,* Southwestern District, Albany, Ga.

HERBERT A. MCCLURE, M.D.,† of Butler, Ala., Health Officer of Choctaw County, has been appointed Health Officer in Lamar County, with headquarters in Vernon; he succeeds DR. DANIEL R. BROWN, resigned.

DR. ROBERT K. MCINTOSH, JR., of Tahlequah, Okla., has been appointed Health Officer of Cherokee County.

DR. HUGH H. MONROE, of Lindsay, Okla., has been appointed Health Officer of Garvin County.

JOSEPH W. MOUNTIN, M.D.,* Senior Surgeon, U. S. Public Health Service, Washington, D. C., has been relieved from his assignment in charge of public health methods at the National Institute of Health, Bethesda, Md., and detailed as Assistant Surgeon General in charge of the Division of Domestic Quarantine. He succeeds Assistant Surgeon General CLIFFORD E. WALLER,* who has been assigned to duty in the Surgeon General's office in charge of the General Inspection Office.

DR. JAMES O. NALL, of Clay, Ky., has been appointed Health Officer of Lawrence County, with headquarters at Louisa.

JACK B. PORTERFIELD, M.D.,† of Williamsburg, Va., Health Officer of the Peninsula District, has been made Epidemiologist of the Virginia State Department of Health, Richmond, Va.

DR. JAMES C. RUMLEY, of Stigler, Okla., has been appointed Health Officer of Haskell County.

WILLIAM V. SANFORD, M.D.,† of Cleveland, Tenn., for 9 years Director of the Bradley County Health Department, has been appointed Health Commissioner of Chattanooga; he succeeds DR. JOHN W. L. COOPER, who resigned to devote full time to private practice.

DR. HARRY A. SMITH, of Wheeling, W. Va., has been appointed Health Officer of Wheeling and Ohio County.

DR. WILLIAM D. SMITH, of Senatobia, Miss., has been appointed Health Officer of Tate County, succeeding DR. JOHN SIDNEY EASON, of Coldwater.

DR. DAVID B. SNELLING, of Tuscumbia, Ala., formerly Assistant to the Health Officer of Colbert County, has been appointed Health Officer of Choctaw County; he succeeds HERBERT A. MCCLURE, M.D.,† of Butler, who has taken a similar position in Lamar County.

DR. JOHN E. TATE, of Ripley, Miss., has been appointed Health Officer of Tippah County, for a 2 year term.

DR. STUART P. VANDIVIERE, of Franklin, N. C., has been placed in charge of the Baldwin County, Ga., Health Department.

DR. JOHN G. WELCH, of Port Neches, Tex., has been appointed Health Officer of Beaumont.

DR. ROBERT K. WILSON, of Dothan, Ala., has been appointed Health Officer of Pickens County, succeeding DR. HORACE C. HUNT, of Livingston, who resigned to enter private practice of medicine.

### Western States

JACQUES P. GRAY, M.D., M.P.H.,* who for two years has been Director of Public Welfare in the City and County of San Francisco, Calif., has resigned, effective August 15, to become lecturer in public health in the

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† Member A.P.H.A.

Department of Hygiene, University of California, Berkeley. Dr. Gray will be responsible for courses in the Department organized by the late Dr. John Force on child health, community control of the communicable diseases, elementary public health, and advanced epidemiology.

EDWARD E. HAMER, M.D., of Carson City, formerly States Health Officer of Nevada, was recently reappointed to that position.

WILLIAM M. MCKAY, M.D.,† of Salt Lake City, Utah, Director of the Division of Communicable Disease Control of the Utah State Board of Health, has been made Acting State Health Commissioner, while JOHN L. JONES, M.D.,* the Health Commissioner, is conducting a survey of occupational disease in the state.

DR. EUGENE W. ROCKEY, of Portland, Ore., has been appointed Chief of Staff of the Industrial Accident Commission, succeeding DR. LOUIS P. GAMBEE. Dr. Rockey held this position once before.

DR. KENNETH M. SODERSTROM, of Seattle, Wash., was recently appointed to the staff of the Division of Epidemiology in the Washington State Department of Health, in charge of tuberculosis control, a newly created position.

DR. RICHARD H. WILCOX, of Pierre, S. D., has been appointed Health Officer of Umatilla County, to succeed ALFRED H. MACLAREN, M.D.,† who will enter private practice in California.

### Canada

DR. DUNCAN GRAHAM, of Toronto, Ont., was named President-Elect of the Canadian Medical Association, at the recent annual meeting in Montreal.

DR. MORLEY S. LOUGHEED, City Bacteriologist for several years, has been appointed Health Officer of Winnipeg, Man., to succeed DR. ALEXANDER J. DOUGLAS, M.D.,* who is retiring after nearly 40 years of service.

DR. HERBERT C. GEORGE, of Regina, Sask., has been appointed Medical Director of Cancer Services for the Saskatchewan Cancer Commission and Director of the Regina Cancer Clinic; and DR. ALLAN W. BLAIR, of Toronto, Ont., has been made Supervisor of Radiotherapy for the Commission and Radiotherapist of the Clinic.

### Foreign

LEONIDES ANDREU ALMAZAN, M.D.,† of Mexico City, has resigned as Chief of the Department of Public Health in Mexico, and has been succeeded by DR. Y GRAL. JOSE SIUROB, who was his predecessor for several years in the same position.

### DEATHS

ERNEST C. DICKSON, M.D.,* of San Francisco, Calif., Professor of Public Health and Preventive Medicine at Stanford University Medical School, died August 23.

JOSEPH E. GERMAIN, D.P.H.,† Director of Rimouski Health Unit, Rimouski, Que., Canada, died recently, at the age of 53.

DR. HAROLD RYPINS, Secretary of the New York State Board of Medical Examiners, Albany, N. Y., died recently.

F. E. TROTTER, M.D.,† President and Executive Officer, Territorial Board of Health, Honolulu, T. H., died recently.

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## CONFERENCES AND DATES

American Association for the Advancement of Science. Annual Meeting —Columbus, Ohio, December 27, 1939–January 2, 1940.

American Clinical and Climatological Association. Saranac Lake, N. Y. October 9–11.

American College of Surgeons. Philadelphia, Pa. October 16–20.

American Education Week — 1939. Theme, "Education for the American Way of Life." November 6–11.

**American Public Health Association. 68th Annual Meeting. Hotel William Penn, Pittsburgh, Pa. October 17–20.**

American Public Welfare Association. Round Table and Annual Meeting. Washington, D. C. December 6–10.

American Public Works Association. Pittsburgh, Pa. October 9–11.

American School Health Association. Hotel William Penn, Pittsburgh, Pa. October 16–20.

American Statistical Association—Annual Meeting. Philadelphia, Pa. December 27–30.

American Water Works Association:

- Four States Section. Berkshire Hotel, Reading, Pa. October 5–6.
- Missouri Valley Section. Hotel President, Kansas City, Mo. October 11–13.
- West Virginia Section. Morgan Hotel, Morgantown, W. Va. October 12–14.
- Southwest Section. Adolphus Hotel, Dallas, Tex. October 16–19.
- New Jersey Section. Claridge Hotel, Atlantic City, N. J. October 20–21.
- California Section. Fairmont Hotel, San Francisco, Calif. October 25–28.
- Wisconsin Section. Hotel Raulf, Oshkosh, Wis. October 30–November 1.
- New York State Section. Hotel Utica, Utica, N. Y. November 2–3.
- North Carolina Section. Hotel Charlotte, Charlotte, N. C. November 6–8.

Association of American Medical Colleges. Cincinnati, Ohio. October 23–25.

Association of Women in Public Health. Century Club, Pittsburgh, Pa. October 16.

Central Society for Clinical Research. Chicago, Ill. November 3–4.

Civil Service Assembly of the United States and Canada. San Francisco, Calif. October 16–20.

Civil Service Assembly—Western Regional Conference. San Francisco, Calif. October 17.

Colorado Public Health Association. Colorado Springs, Colo. October 3–4.

Convention for the Revision of the Pharmacopoeia of the United States. Washington, D. C. May 14, 1940.

Directors of Local Health Services Conference. Hotel Roosevelt, Pittsburgh, Pa. October 16.

First Annual Symposium for Industrial Nurses. Presented by the Department of Public Health of the State of Illinois. University of Illinois Medical School, Chicago, Ill. October 26–28.

Florida Public Health Association. Jacksonville, Fla. December 7–9.

Health Education Institute—American Public Health Association, Annual Meeting. Hotel William Penn, Pittsburgh, Pa. October 15–17.

International Association of Milk Sanitarians, Inc.—Annual Meeting. New Hotel Mayflower, Jacksonville, Fla. October 25–27.

International Heating and Ventilating Exposition—Sixth. Under auspices

of the American Society of Heating and Ventilating Engineers, and coinciding with its 46th Annual Meeting. Lakeside Hall, Cleveland, Ohio. January 22–26, 1940.

International Society of Medical Health Officers. Hotel William Penn, Pittsburgh, Pa. October 16.

Michigan Public Health Association. Grand Rapids, Mich. November 8–10.

Mississippi Public Health Association. Jackson, Miss. December 6–8.

Municipal Public Health Engineers Conference. Hotel William Penn, Pittsburgh, Pa. October 16.

National Association for Nursery Education — Biennial Meeting. Hotel Pennsylvania, New York, N. Y. October 25–28.

National Association of Housing Officials. New Orleans, La. November 22–24.

National Association of Public School Business Officials. Cincinnati, Ohio. October 16–20.

National Association of Sanitarians. Oakland, Calif. December.

National Hearing Week—12th. Announced by the American Society for the Hard of Hearing. October 22–28.

National Organization for Public Health Nursing. Hotel William Penn. Pittsburgh, Pa. October 15–16.

National Pest Control Association—7th Convention. Hotel Pennsylvania, New York, N. Y. October 23–25.

National Recreation Congress — 24th. Boston, Mass. October 9–13.

National Safety Congress and Exposition—28th. Atlantic City, N. J. October 16–20.

National Society for the Prevention of Blindness. Annual Conference. Hotel Astor, New York, N. Y. October 26–28.

National Warm Air Heating and Air Conditioning Association. Cleveland, Ohio. January 22–26, 1940.

New Jersey Health and Sanitary Association — 65th Annual Meeting. Berkeley-Carteret Hotel, Asbury Park, N. J. November 24–25.

New Mexico Public Health Association. LaFonda Hotel, Santa Fe, N. M. October 25–28.

New York State Association of Public Health Laboratories. Mid-Year Meeting. State Laboratory, Albany, N. Y. November 3.

New York State Sewage Works Association. Olean House, Olean, N. Y. October 6–7.

Pennsylvania Public Health Association—Meeting with American Public Health Association. Hotel William Penn, Pittsburgh, Pa. October 18.

Southern Branch, American Public Health Association — 8th Annual Meeting. Memphis, Tenn. November 21–22.

Southern California Public Health Association. Long Beach, Calif. January 24, 1940.

Southern Medical Association. Memphis, Tenn. November 21–24.

State Laboratory Directors Conference. Hotel William Penn, Pittsburgh, Pa. October 16.

State Sanitary Engineers Conference. Hotel William Penn, Pittsburgh, Pa. October 16–17.

Texas Public Health Association. Galveston, Tex. October 2–4.

Tri-State Food & Health Officials. Hotel William Penn, Pittsburgh, Pa. October 18.

West Virginia Public Health Association. Hotel Fairmont, Fairmont, W. Va. November 6–8.

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